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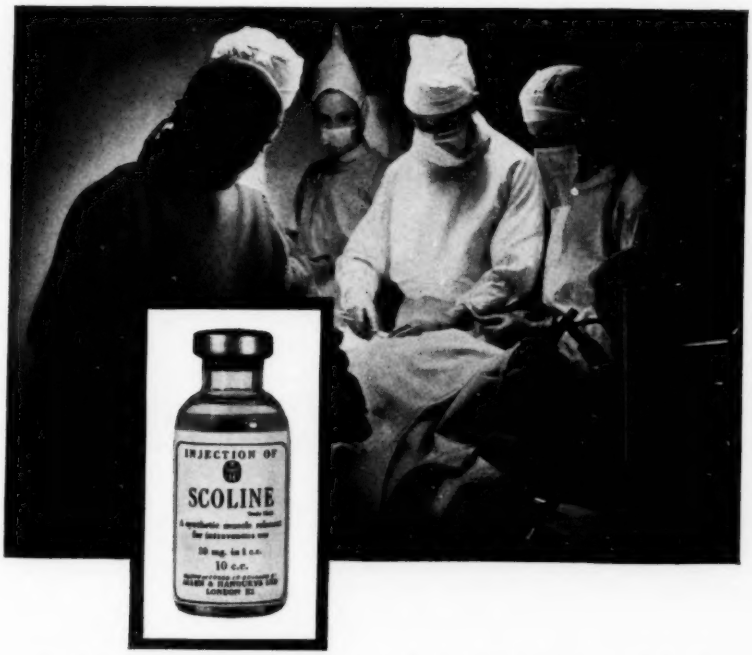


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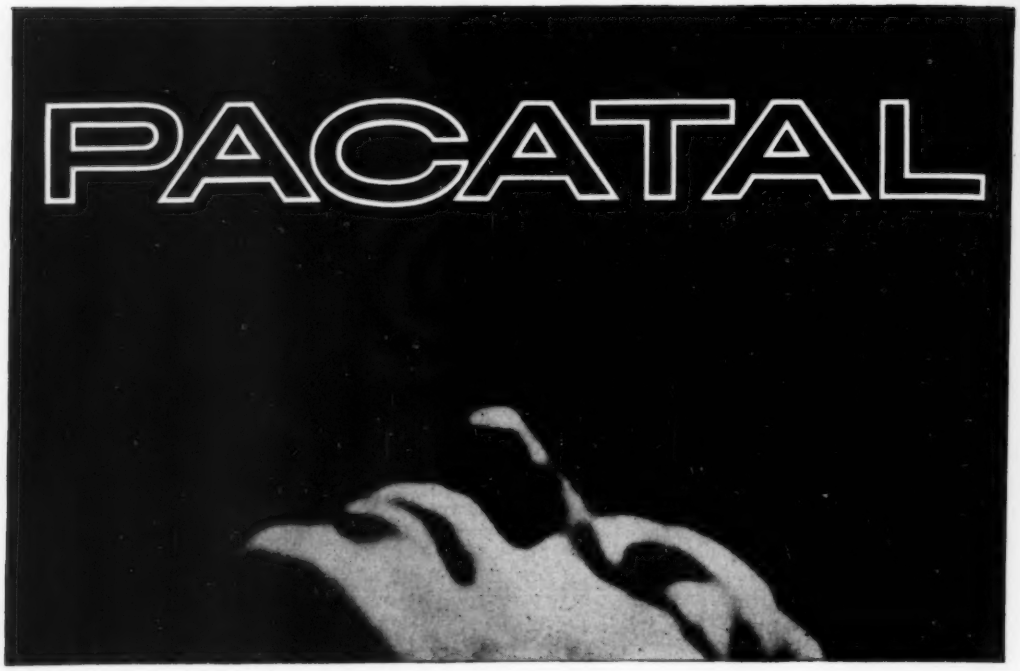

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# BRITISH JOURNAL OF ANAESTHESIA

VOL. XXIX No. 11 NOVEMBER 1957

## EDITORIAL

### SILVER JUBILEE OF THE ASSOCIATION OF ANAESTHETISTS

OUR greetings go to the Association of Anaesthetists of Great Britain and Ireland, which this year celebrates the twenty-fifth anniversary of its foundation. Its achievements have been many and great and there is no anaesthetist in this country who does not owe to that body a very great deal indeed. The status of the Consultant Anaesthetist in the Health Service was achieved not only by the increasing complexity of the techniques which he had to use and scope of knowledge and experience which they demanded, but also by a steady build-up of the anaesthetist over the past twenty-five years by such steps as the introduction by the Conjoint Board of the Diploma in Anaesthetics and the Foundation of the Faculty within the Royal College of Surgeons. In these advances the Association played a leading role. Through its President at the time, Dr. Archibald Marston, it was also instrumental in the formation of the B.M.A. group of Anaesthetists. The launching of the journal *Anaesthesia* was a considerable landmark in the history of the Association and while many may regret the necessity for two journals, none can doubt that both have maintained their own individual approaches and will continue to keep the anaesthetic research of this country before the eyes of the world.

We wish the Association a future as strong, useful and progressive as its first quarter century.

### A NEW JOURNAL—A NEW DEPARTMENT

EXPANSION and progress are not necessarily coterminous. But the launching of a new journal, *Acta Anaesthesiologica Scandinavica*, cannot but be welcomed. The Scandinavian countries have contributed and are contributing much to our specialty and this forum for their original work has been long awaited. Under the editorship of our friend Dr. Eric Nilsson of Lund we can be sure of a high standard. The journal is published in English in an attractive format with a clear and dignified type. It is a worthy publication of that progressive and forward-looking region of Europe.

A further example of expansion which must also be regarded as progress is the institution of a Department of Anaesthetics in Queen's University, Belfast. Dr. John W. Dundee has been elected as the first Director of the Department. The University Departments of Anaesthetics have played a leading part in the advance of anaesthesia over the last decade. Our day-to-day practice has become more rational and less empirical as a result of their fundamental researches. More and more light has been shed on the action of drugs, the problems of pulmonary ventilation in all their aspects and on the reaction of patients to anaesthesia and surgery. This latest centre will no doubt help to maintain the pace of exploration and the application of the new knowledge for the benefit of our patients.

*Brit. J. Anaesth.* (1957), **29**, 486

# INFRARED ANALYSIS FOR THE ESTIMATION OF CARBON DIOXIDE IN THE PRESENCE OF NITROUS OXIDE

BY

E. A. COOPER

*Department of Anaesthetics, King's College, Newcastle upon Tyne*

## PART I

### THE EFFECT OF NITROUS OXIDE

RAMWELL (1957a, b) has shown that anaesthetic gases may cause serious errors in the estimation of carbon dioxide by the technique of infrared analysis, even though these gases be used as filters to correct for their own ability to absorb radiation. The errors arise from an action of the molecules of the diluent gas upon those of carbon dioxide causing alteration in its infrared absorption spectrum (pressure broadening effect). The shape of the curve relating absorption to wavelength, the amplitude of the peaks and even the wavelength at which they occur may variously be altered (Foley, 1946; Coggeshall and Saier, 1947). Owing to such complexity, it is not practicable to make allowance for the effect by theoretical methods. Ramwell has suggested that the errors may be avoided if, in addition to the normal precautions, calibration curves are established for the percentage of carbon dioxide in a definite mixture of anaesthetic gases having the same composition as that expired by the patient. He has stated "care must be taken to adhere to the composition of the diluent gases of the calibration curve during the period of anaesthesia, which means that rebreathing of the gas mixture must be avoided". Coggeshall and Saier have, however, shown that the optical density of a mixture of carbon dioxide and a diluent bears a power law relationship to the partial pressure of the diluent (approximately proportional to pressure$^{0.68}$). It seems likely, therefore, that if the partial pressure of a diluent gas is large, small changes in it will have little influence on the pressure broadening effect. Thus, it is likely that if the concentration of nitrous oxide in a gas mixture is high, its pressure broadening effect may be large but fairly constant over a moderate range of change of concentration. The relevant results in Ramwell's papers support this conclusion. It was therefore decided to make a calibration curve for the estimation of carbon dioxide in pure nitrous oxide, and subsequently to enquire whether such a curve could be modified for the measurement of the percentage of carbon dioxide in various mixtures of nitrous oxide and oxygen.

#### METHOD

The infrared gas analyzer was a standard model SC/P made by the Infra Red Development Co. Mixtures were made from dry gases. The filter chamber was always full of nitrous oxide at atmospheric pressure and was regularly flushed. Zero was set with oxygen in the sample chamber and the gain control was adjusted to cause a constant arbitrary deflection with a standard calibrating mixture of 5.22 per cent carbon dioxide in oxygen.

Zero adjustment and calibration were checked immediately before and after the analysis of each sample. Only one measurement was made on each mixture, but this was an estimated mean over a period of about half a minute. Experimental mixtures were made accurately in a gas burette.

#### RESULTS

Experiments were carried out on twenty-two mixtures of carbon dioxide (0 to 9 per cent) in nitrous oxide. The solid circles in figure 1 represent the percentages of carbon dioxide in the mixture made in the gas burette plotted against

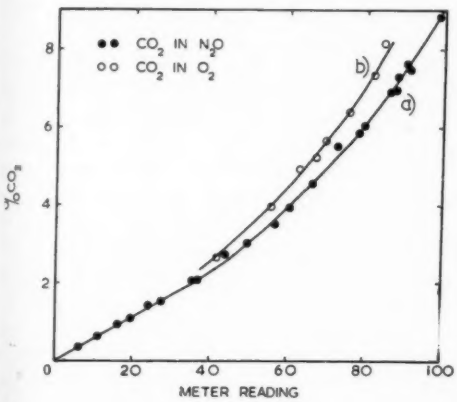


FIG. 1

The known percentage of carbon dioxide in the gas mixtures is plotted against the reading of the infrared analyzer. The curves have been fitted by eye.

the reading of the infrared gas analyzer. The results showed a satisfactory relationship between the percentage of carbon dioxide in nitrous oxide and the reading of the galvanometer of the infrared analyzer. The curve (a) was drawn by eye to fit the points. Experiments were also carried out on eight mixtures of carbon dioxide (2.5 to 8.5 per cent) in oxygen. The results are shown by the hollow circles in figure 1. The curve (b) was also drawn by eye.

Experiments were carried out on fourteen mixtures in which the percentage of carbon dioxide was between 2.5 and 8. The percentage of nitrous oxide in the diluent mixture was between 34 and 80. The experimental results are not shown here, but will be discussed in detail below. All but one fell in the area bounded by the two curves of figure 1. In general, the points for mixtures of high nitrous oxide content fell only slightly above the nitrous oxide curve (a), while the points for mixtures containing less nitrous oxide fell farther above.

## DISCUSSION

The lack of agreement between the results for carbon dioxide in nitrous oxide and in oxygen (fig. 1) confirms Ramwell's findings though the discrepancy is not so great. He found that 5 per cent carbon dioxide in nitrous oxide caused a deflection equal to that produced by 6.2 per cent carbon dioxide in oxygen. In the present experiments the deflection due to 5 per cent carbon dioxide in nitrous oxide was equal to that produced by a 5.77 per cent mixture in oxygen.

The two standard reference mixtures used for setting up of the infrared analyzer were pure oxygen and 5.22 per cent carbon dioxide in oxygen. It can therefore be said that once the calibration curve is established for the estimation of carbon dioxide in nitrous oxide, the subsequent use of the analyzer does not entail use of standard mixtures of these latter two gases. The difficulties of obtaining and analyzing mixtures of carbon dioxide in nitrous oxide may thus be avoided. The purpose of the standard mixture of carbon dioxide in oxygen is the establishment of a repeatable setting of the analyzer. The validity of the calibration curve for carbon dioxide in nitrous oxide depends on this. The initial standard reference mixture of carbon dioxide in oxygen will, however, eventually become exhausted. It is therefore necessary to establish the relationship between the percentage of carbon dioxide in oxygen and the galvanometer deflection which will ensure the repeatability of the setting of the analyzer. The curve (b) of figure 1 defines this relationship. Thus, in the future, if a standard mixture of $x$ per cent carbon dioxide in oxygen were available, the analyzer could be adjusted to give the deflection which corresponded to $x$ per cent on curve (b). Curve (a) would then be valid for the estimation of carbon dioxide in nitrous oxide.

The results of the experiments on carbon dioxide in mixtures of nitrous oxide and oxygen showed that, as expected, when the percentage of nitrous oxide was high the points fell close to the curve for carbon dioxide in nitrous oxide. The error was therefore assessed which would arise from the use of this curve (a) in estimating the percentage of carbon dioxide in anaesthetic mixtures of nitrous oxide and oxygen. Figure 2 shows this error plotted against the percentage of nitrous oxide in the diluent gas. It is expressed as a percentage of the actual quantity of carbon dioxide present (which was known from the readings of the gas burette in which the mixture was made).

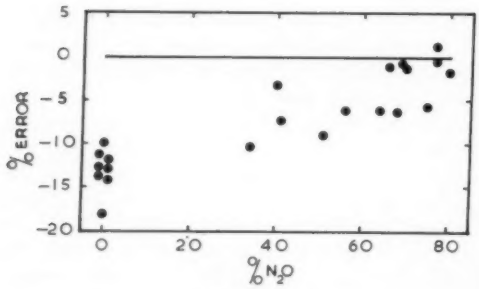


FIG. 2

The ordinate represents the error entailed in using the calibration curve of carbon dioxide in nitrous oxide for the estimation of carbon dioxide in mixtures of nitrous oxide and oxygen. The error is expressed as a percentage of the true value for carbon dioxide. The abscissa represents the amount of nitrous oxide in the diluent mixture.

The figure shows an increase in the error of underestimation as the percentage of nitrous oxide fell, but the error in the range of nitrous oxide percentage between 60 and 80 was not large. When the percentage of nitrous oxide was between 71 and 80 the mean error was − 1.7 per cent, when between 61 and 70 the mean error was − 3.16 per cent. The mean error for the points of these two groups was −2.51 per cent. Thus there was only a slight systematic error entailed in the assumption that diluent mixtures containing 60 to 80 per cent nitrous oxide in oxygen had a common effect on the estimated percentage of carbon dioxide. It was therefore concluded that it would be reasonable to estimate carbon dioxide in such a mixture by the use of the calibration curve of figure 1 for carbon dioxide in nitrous oxide, after the appropriate modification of the ordinate scale by 2.5 per cent. In order to check the validity of this conclusion, the method was applied to the results obtained for the nine relevant gas mixtures. Table I shows the mean error and range of errors. They are expressed both as percentage of correct value and as percentage of carbon dioxide. Such errors include that due to the use of the gas burette, that due to the use of the infrared gas analyzer, and that due to the use of the approximation. The first two of these are also entailed in the establishment of the calibration curve for carbon dioxide in nitrous oxide, and appear as the deviation of the experimental points about it in figure 1. These deviations have been similarly calculated as errors and their means and ranges are also shown in table I. The results for the estimation of carbon dioxide in nitrous oxide were similar to those for its estimation in mixtures of nitrous oxide and oxygen. Thus the additional overall error due to the approximation was negligible. Since it was shown above that the systematic error due to variation of nitrous oxide between 60 and 80 per cent was slight, it was therefore concluded that a single calibration curve, based on that for carbon dioxide in nitrous oxide, could be used satisfactorily for the estimation of carbon dioxide in mixtures of nitrous oxide, 60 to 80 per cent, and oxygen. It is considered that this general conclusion will also be applicable to other infrared gas analyzers. Each apparatus, however, has its own specific behaviour and it would be necessary to construct the corresponding calibration curve from experimental findings.

Ramwell has shown that the behaviour of nitrogen, in the present context, is more like that of oxygen than that of nitrous oxide. Coggeshall and Saier (1947) have also shown similarity in the effects of nitrogen and oxygen on the absorption of infrared radiation by carbon dioxide. The process of denitrogenation of the body will not be discussed here, but Hamilton and Eastwood (1955) have shown that one minute of normal ventilation through the commonly used anaesthetic circuits, with a pure oxygen flow of not less

TABLE I

| | Error % correct value | Error % $CO_2$ |
|---|---|---|
| $CO_2$ in mixtures of $N_2O$ and $O_2$ (9 points) | Mean ±2.32%<br>Range −3.75 to +3.85% | Mean ±0.099%<br>Range −0.164 to +0.240% |
| $CO_2$ in $N_2O$ (14 points) | Mean ±2.06%<br>Range −2.87 to +8.34% | Mean ±0.106%<br>Range −0.190 to +0.271% |

than 4 l./min, suffices to reduce the percentage of nitrogen in late expired gas to 20 or less. This percentage is approximately halved during the next minute. It is therefore likely that following alveolar ventilation equivalent to about two minutes of normal respiration, with nitrous oxide 70 to 80 per cent in oxygen, the expired gas will have a composition to which the method discussed above for the estimation of carbon dioxide is applicable.

## Part II

### THE EFFECT OF WATER VAPOUR

The presence of water vapour in a sample of expired gas would be expected to have some effect on heat absorption. The total effect would be the resultant of three possible mechanisms: simple dilution of the carbon dioxide; pressure-broadening effect on the absorption spectrum of carbon dioxide; absorption of heat by the water molecules themselves. Collier, Affeldt and Farr (1955), using a 4 per cent mixture of carbon dioxide in air, reported that the sum of these effects was negligible when the gas was saturated at 37°C. Ramwell (1957b) reported that the variation in the estimate of 5 per cent carbon dioxide due to saturation was usually not greater than 0.03 per cent of carbon dioxide. The composition of the diluent gas and the temperature of saturation were not specified. Investigation is reported here of the effect of saturation at room temperature of various mixtures of carbon dioxide, nitrous oxide and oxygen.

#### METHOD

The gas mixtures were made up in large polythene bags immediately before use. Their composition was roughly controlled by the use of rotameter flow metres. When desired, the gases were saturated by bubbling slowly through acidulated water in a 200 ml flask which was continually shaken. Its temperature was maintained at or slightly above that of the room (16°C). The samples were passed through the flask and the analyzer until a steady reading was obtained over a period of two minutes. Each mixture was passed through the analyzer at least four times (twice dry and twice saturated).

#### RESULTS

Four gas mixtures were used whose approximate compositions are shown in table II. Fourteen pairs of readings were made and the final column shows the difference in the deflections due to the

Table II

| Approximate % $CO_2$ | Approximate % gas in diluent | | Deflection (dry) minus deflection (wet) |
|---|---|---|---|
| | $N_2O$ | $O_2$ | |
| 7 | 100 | | 0 |
| | | | 0 |
| | | | −0.4 |
| 5 | | 100 | 0 |
| | | | 0 |
| | | | 0 |
| | | | 0 |
| | | | +0.5 |
| | | | +0.3 |
| | | | +0.2 |
| 3 | 45 | 55 | 0 |
| | | | +0.2 |
| 2 | 30 | 70 | 0 |
| | | | 0 |

dry and wet gases, expressed always as deflection (dry) minus deflection (wet). They were slight and irregular. The greatest difference was equivalent to an error in estimation of less than 0.03 per cent of carbon dioxide.

The presence of water vapour in the gases at room temperature was thus shown not to affect the reading of the galvanometer of the infrared analyzer. Therefore, the interpretation of this reading by the standard calibration curve gives the percentage of carbon dioxide in the dry gases and subsequent correction must be made to this figure if it is desired to describe the percentage of carbon dioxide in moist expired gases.


## SUMMARY

The findings of previous workers have been confirmed that the presence of nitrous oxide leads to errors in the estimation by infrared analysis of carbon dioxide in gas mixtures.

Evidence is presented that, once the calibration curve is established, an infrared gas analyzer can be used for the estimation of carbon dioxide in nitrous oxide without subsequent reference to standard mixtures of these gases.

It is suggested that, with appropriate modification of the calibration curve, this method can be used with satisfactory accuracy in the estimation of carbon dioxide in mixtures of nitrous oxide, 60 to 80 per cent, and oxygen. Within these limits, it is not necessary to know precisely the percentage of nitrous oxide. The analyzer can thus be used with many standard anaesthetic techniques including those involving partial rebreathing.

Evidence is also presented on the effect of saturation at room temperature on the estimation of carbon dioxide in various gas mixtures.



## ACKNOWLEDGMENTS

I wish to thank Professor E. A. Pask and Mr. L. Molyneux for their very valuable help throughout all stages of this work.


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# PARTIAL PRESSURES OF OXYGEN IN THE MAGILL ATTACHMENT*

BY

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THE fresh gas flow rates commonly advised for anaesthesia with nitrous oxide, oxygen and ether (using the Magill attachment) are of the order of 5 to 8 litres per minute. It has been shown by Harrison and Jones (1955) that with a fresh gas flow rate of 8 litres per minute, a considerable accumulation of carbon dioxide occurs when normal subjects breathe from the Magill attachment and clinical evidence suggests that this state of affairs may obtain during anaesthesia also (Jones and Harrison, 1957).

While the rebreathing of any expired air other than the inspiratory deadspace gas (which has the same composition as the ambient atmosphere) will tend to lead to $CO_2$ accumulation in the breathing apparatus and the subject, it should also be remembered that exhaled alveolar air has a lower oxygen content than inhaled air.

The addition of any gas with little or no oxygen content to inhaled air will, in accordance with Dalton's law of partial pressures, lead to an overall diminution of the oxygen content of the inhaled air. This has been confirmed in practice by Crowley et al. (1948) and by Schwartz et al. (1953).

The possible untoward effects of such anoxic anoxia have been discussed by Courville (1939). The majority of the cases he described were subjected to inhaled mixtures with oxygen contents held deliberately at very low concentrations (10 per cent or even less). These patients were also rebreathing unspecified volumes of expired air and it is not unlikely that this rebreathing technique, of itself, further lowered the partial pressure of oxygen in the patient's alveolar air.

In the use of the Magill attachment, the volume of exhaled air which is rebreathed varies according to the respiratory ventilating volumes of the patient and the rate of flow of fresh gas into the attachment. The arbitrary selection of an average flow rate of fresh gas of 8 litres per minute is probably based upon figures for respiratory rate and depth usually quoted in textbooks (Comroe et al., 1955). If the subject does not conform to average and has a respiratory ventilation greater than the fresh gas minute volume, the volume of rebreathed expired air may become considerable.

The estimation of the carbon dioxide content of anaesthetic atmospheres is not simple, but the paramagnetic properties of oxygen are specific and can be employed to give rapid and accurate estimates of the oxygen content of inhaled atmospheres. It seemed to us that a study of the oxygen partial pressures in the Magill attachment might be of value in demonstrating any dilution effect from rebreathing alveolar air.

## EXPERIMENT

In a series of 16 patients (9 of them males) ranging in age from 12 to 77 years (mean 38 years) and in weight from 80 to 220 pounds (mean 146 lb, i.e. 66 kg) oxygen partial pressures were measured during extra-abdominal operations under thiopentone-nitrous oxide-oxygen-ether anaesthesia using a Boyle's type anaesthetic machine and a Magill attachment. All patients received the ordinary forms of premedication with morphine (or pethidine) and atropine in doses scaled roughly to weight and general condition. No anaesthetic agent other than nitrous oxide and ether was administered by inhalation nor was anything other than 2.5 per cent solution of thiopentone administered intravenously. The dosage of thiopentone varied from 180 mg to 800 mg (mean 465 mg) and the total quantities of ether used in each of the 30-minute observa-

* The cost of this work was defrayed by a Research Grant from the South African Council for Scientific and Industrial Research.

tion periods varied from 1 to 4 ounces (mean 2.6 oz, i.e. 65 ml). Fresh gas flow rates of 6 litres per minute of nitrous oxide and 2 litres per minute of oxygen were constant for all patients. Respiratory rates during anaesthesia varied between 14 and 40 per minute.

Gas samples for analysis were drawn from two points (fig. 1). Point A was just proximal to the reservoir balloon mount and point B was at the face piece. Sampling was undertaken at regular intervals, but in planning this study it was decided to restrict this report to the results of samples taken during the 20th minute and during the 30th minute of the anaesthetic. By this time the bulk of the nitrogen has been eliminated and the patient is approaching a steady state although the table of results shows (see table I) that there is still a drift between the 20th and the 30th minutes.

A Pauling oxygen meter (Model C) was used to analyze gas samples. The samples were drawn continuously through the sampling chamber of the meter over a period of 1 minute and the reading was taken at the end of the minute of sampling. Sample volumes were sufficiently large to flush the analyzing chamber completely. The oxygen meter was calibrated against various mixtures of oxygen and air or oxygen and nitrogen. The composition of these mixtures was determined by triplicate analysis using a Haldane gas analysis apparatus. A calibration curve was constructed and all readings of the meter were corrected by reference to this curve. At the beginning and at the end of each experiment the meter was checked for zero and full scale deflections by flushing it first with nitrous oxide and then with oxygen. The latter reading was further checked against the barometric pressure.

## RESULTS

The results are set out in table I and are expressed both as mm Hg $pO_2$ and as $O_2$ percentage of the gas mixture (at atmospheric temperature and pressure and saturated with water vapour).

It will be seen that the mean values at the sampling points increase slightly between the 20th and 30th minutes of anaesthesia. There are at least two possible explanations for this drift. The more likely is the fall in the partial pressure of ether. The loss of heat with the evaporation of ether leads to cooling of the liquid ether remaining in the vaporizer and in turn this leads

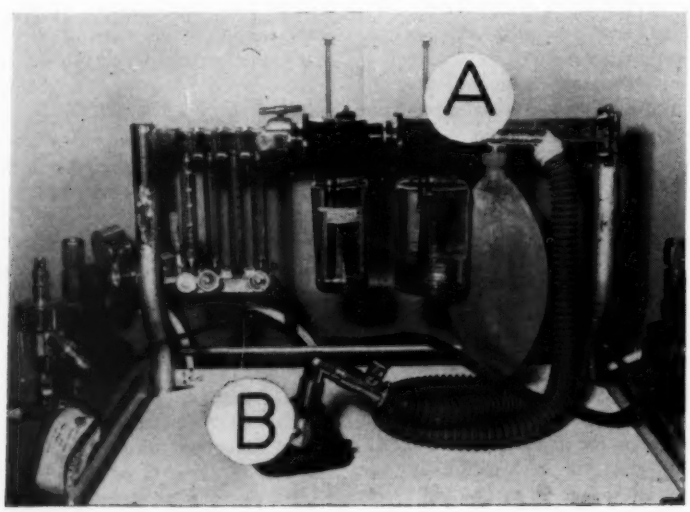


FIG. 1

The type of anaesthetic machine used in the experiments, showing the two points of sampling.

TABLE I

| Patient No. | Age in years | Weight in pounds | Barometric pressure mm Hg | Oxygen at A | | | | Oxygen at B | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | At 20th min | | At 30th min | | At 20th min | | At 30th min | |
| | | | | $pO_2$ mm Hg | $O_2$ vols per cent | $pO_2$ mm Hg | $O_2$ vols per cent | $pO_2$ mm Hg | $O_2$ vols per cent | $pO_2$ mm Hg | $O_2$ vols per cent |
| 1 | 59 | 190 | 771 | 212 | 27.5 | 213 | 27.6 | 187 | 24.2 | 190 | 24.6 |
| 2 | 44 | 190 | 771 | 190 | 24.6 | 195 | 25.3 | 160 | 20.8 | 173 | 22.4 |
| 3 | 45 | 110 | 760 | 187 | 24.6 | 210 | 27.6 | 157 | 20.7 | 167 | 22.0 |
| 4 | 16 | 130 | 760 | 194 | 25.5 | 214 | 28.2 | 177 | 23.3 | 190 | 25.0 |
| 5 | 35 | 140 | 760 | 200 | 26.3 | 199 | 26.2 | 190 | 25.0 | 178 | 23.4 |
| 6 | 26 | 200 | 760 | 205 | 27.0 | 214 | 28.2 | 177 | 23.3 | 194 | 25.5 |
| 7 | 35 | 110 | 756 | 190 | 25.1 | 184 | 24.3 | 178 | 23.5 | 174 | 23.0 |
| 8 | 77 | 170 | 756 | 190 | 25.1 | 197 | 26.1 | 178 | 23.5 | 190 | 25.1 |
| 9 | 33 | 110 | 756 | 199 | 26.3 | 203 | 26.9 | 173 | 22.9 | 182 | 24.1 |
| 10 | 22 | 190 | 760 | 194 | 25.5 | 194 | 25.5 | 174 | 22.9 | 169 | 22.2 |
| 11 | 34 | 120 | 771 | 184 | 23.8 | 190 | 24.6 | 169 | 21.9 | 171 | 22.2 |
| 12 | 30 | 150 | 771 | 199 | 25.8 | 197 | 25.6 | 190 | 24.6 | 184 | 23.9 |
| 13 | 42 | 120 | 771 | 209 | 27.1 | 204 | 26.5 | 195 | 25.2 | 184 | 23.9 |
| 14 | 56 | 130 | 759 | 173 | 22.8 | 178 | 23.4 | 163 | 21.8 | 167 | 22.0 |
| 15 | 12 | 80 | 759 | 190 | 25.0 | 190 | 25.0 | 178 | 23.7 | 178 | 23.7 |
| 16 | 49 | 220 | 771 | 191 | 24.8 | 190 | 24.6 | 174 | 22.6 | 174 | 22.6 |
| Mean | 38 | 146 | 764 | 188 | 25.4 | 194 | 26.0 | 176 | 23.1 | 179 | 23.5 |
| S.D. | | | | | ±1.3 | | ±1.4 | | ±1.3 | | ±1.2 |

to a reduction in the vapour pressure of the ether vapour. The less likely explanation (at this point of time in the anaesthetic) is the continued elimination of nitrogen. The slow accumulation of carbon dioxide cannot be excluded altogether as a cause of this drift (Harrison and Jones, 1955).

The most important fact to note is the fall in the mean value for $pO_2$ between sampling point A and sampling point B. This is graphically shown in figure 2. At the 20th minute the $pO_2$ shows a difference between the sampling points of 12 mm Hg, which is 6 per cent of the $pO_2$ in the gas mixture flowing into the Magill attachment. At the 30th minute the difference in the $pO_2$ at the two sampling points is 15 mm Hg or 8 per cent of the $pO_2$ in the fresh gases from the machine. This fall in the $pO_2$ between point A and point B is due to the diluent effect of exhaled nitrogen, water vapour and carbon dioxide.

Of almost equal interest is the increase in the magnitude of this difference with the passage of time. At the 20th minute the magnitude of the difference is 6 per cent of the $pO_2$ in the fresh gases entering the Magill attachment at A. By the 30th minute, although the mean value for the $pO_2$ of the fresh gas at A has risen, the magnitude of the difference in $pO_2$ between A and B has increased to 8 per cent of the $pO_2$ at A. This effect is also shown graphically in figure 2. As the volume of nitrogen excreted is minimal after the 7th minute (Comroe et al., 1955) (probably about 0.5 per cent or less by volume of each exhalation by the 20th minute) and the vapour pressure of the water vapour is constant at a constant temperature (47 mm Hg at 37° C) the increase in the difference of the $pO_2$ values for the two sampling points from 6 per cent at the 20th minute to 8 per cent at the 30th minute probably represents a slow increase in the partial pressure of carbon dioxide in the inhaled anaesthetic atmosphere due to rebreathing in the Magill attachment.

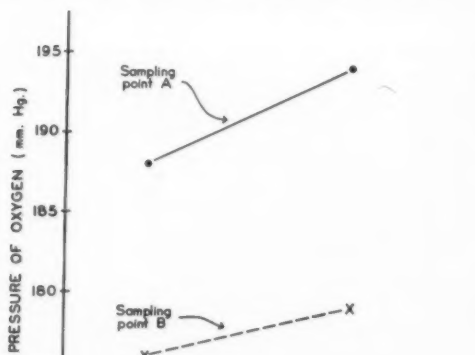


FIG. 2

Partial pressures (mean) of oxygen at the sampling points A and B are here plotted against time to display graphically the increase in the dilution effect with the passage of time.


## SUMMARY

The partial pressures of oxygen in the Magill attachment during clinical anaesthesia in 16 patients have been measured. In addition to the obvious diluent effect of ether and of nitrogen and water vapour added to the gas mixture during the course of the anaesthetic, the behaviour of the oxygen pressures suggests that there is an effect which may be due to the slow accumulation of carbon dioxide in the Magill attachment.


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# ANAESTHETIC POTENCY IN EPIDURAL ANALGESIA

BY

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IN an extensive literature on epidural analgesia there appears to be some difference of opinion as to the intensity of nerve block produced by different drugs, perhaps partly attributable to the fact that many patients are given light narcosis in addition to nerve block, nowadays, which may conceal differences of drug action. In quoting a few of the many authorities, the following drugs are listed according to the original author's assessment of the relative strengths as regards sensory and motor block, independent of duration, although the actual concentrations used by the different observers varied.

Massey Dawkins (1949). Cinchocaine>Procaine.
Bromage (1954a). Lignocaine>Amethocaine>Cinchocaine>Procaine.
Pitkin (1946). Piperocaine>Procaine>Amethocaine.
Lull and Hingson (1945). Piperocaine>Procaine>Butethamine>Amethocaine.
Blundell et al. (1955). Hexylcaine>Lignocaine>Procaine>Amethocaine>Piperocaine.
Crawford (1953). Lignocaine>Hexylcaine>Procaine.
Abajian (1943). Diethoxin>Procaine.

Thus, amethocaine is considered more powerful than procaine by Bromage and weaker than procaine by Pitkin, Blundell et al. and Lull and Hingson. Piperocaine was found to be more powerful than procaine by Pitkin and Lull and Hingson but weaker than procaine by Blundell et al. Other differences of opinion arise over the relative potencies of lignocaine and hexylcaine.

This investigation has been made to ascertain within the limits of restricted clinical trial, the relative potencies of several local anaesthetics in epidural analgesia. Preliminary trials of sacral epidural block (47) and spinal epidural block (53) were carried out to provide experience, using procaine, piperocaine, amethocaine and lignocaine. From a study of this series two principal facts emerged. Firstly, that there appeared to be a far greater variability of response to a given dose than is the case in subarachnoid anaesthesia. For every case of epidural block, twenty intrathecal spinal anaesthetics were administered in the routine work of this department using amethocaine, amylocaine, cinchocaine or procaine. Since uniformity as regards predictability of the extent of block and intensity of relaxation is obtained in upwards of 80 per cent of subarachnoid spinal anaesthesias, the method has formed the basis for comparison with results from epidural anaesthesia. Secondly, anatomical variations accounting for failures were less in the spinal than in the caudal epidurals; it was therefore decided to adopt a standard technique of spinal epidural block and to accumulate comparative series of cases with different anaesthetic solutions. None of the preliminary trial cases have been included, except in the study of toxic reactions. Because of the practical difficulty of delays engendered by the assessment of the results of weak solutions and by the administration of supplementary anaesthesia, the investigation has taken six years to complete.

## TECHNIQUE

Patients were given intravenous premedication of papaveretum 20 mg and hyoscine 0.4 mg for the 10 stone (63 kg) adult with proportional variation for body weight. Reduced dosage was applied also for senility, cachexia and for anaemia with haemoglobin below 70 per cent. The skin over the selected supraspinous ligament was infiltrated with procaine and ephedrine 30 mg and the epidural space identified by release of digital pressure applied to a syringe of normal saline. Nineteen or 20 S. W. G. Luer-Lok short bevel needles were employed. In every case blood pressure charts were made throughout operation. Details of site of spinal puncture and classification of operations appear in table I.

Twelve drugs were used and twenty anaesthetics were conducted for each of thirty-five different solutions and for two techniques with viscous amethocaine. The compositions of the solutions are listed in table II. Structural formulae of all the drugs except Carbocaine and Amplicaine, which are depicted here, were presented by Lloyd (1955). In solutions containing adrenaline the concentration varied from 1/300,000 to 1/150,000, the latter strength being used for laminectomies where an avascular extradural space was advantageous.

TABLE I

*Data of operation, site of spinal puncture and age of the 740 cases.*

| Site of operation | No. of cases |
|---|---|
| Upper limb and chest wall | 12 (mastectomy 8) |
| Stomach ... ... ... | 21 (gastro-enterostomy 15) |
| Liver ... ... ... .. | 14 (hydatid 8) |
| Gall bladder ... ... .. | 12 (cholecystectomy 7) |
| Spleen ... ... ... .. | 6 (hydatid 2) |
| Other upper abdominal ... | 82 (epigastric and umbilical hernia 47) |
| Gut ... ... ... ... | 53 (Appendicectomy 35) |
| Uterus and ovaries ... | 25 (hysterectomy 7) |
| Testis and tunica ... ... | 21 (hydrocele 11) |
| Ureters ... ... ... | 49 (transplantation 25) |
| Bladder ... ... ... | 77 (prostatectomy 41, cystolithotomy 24) |
| Other lower abdominal ... | 204 (inguinal hernia 186) |
| Kidney ... ... ... | 23 (nephrolithotomy 9) |
| Spine ... ... ... ... | 60 (laminectomy 46) |
| Other ... ... ... ... | 7 |
| Lower extremities ... ... | 42 |
| Perineal and gynaecological | 32 |

| Site of interspinous puncture | No. of cases |
|---|---|
| T 3 | 2 |
| T 4 | 5 |
| T 5 | 2 |
| T 6 | 5 |
| T 7 | 2 |
| T 8 | 5 |
| T 9 | 11 |
| T 10 | 29 |
| T 11 | 15 |
| T 12 | 58 |
| L 1 | 131 |
| L 2 | 156 |
| L 3 | 228 |
| L 4 | 91 |

Average age of patients 40.7 years.
Age range from 14 to 80 years.

ASSESSMENT OF RESULTS

Tests of the extent of motor loss, analgesia and hypalgesia were made after injection until no further block developed. Sympathetic block was not awaited prior to commencement of surgery. The extent of hypalgesia was noted at both upper and lower levels as it was found easier to identify the site of pain response in moving from hypalgesic areas to those of normal sensation. Whilst judgment of hypalgesia to pin prick with allowance for apprehensive exaggeration, stoical indifference, ignorance and lack of co-operation is largely arbitrary and personal, every attempt was made to categorize the anaesthesia before incision. Patients were finally classified in the five following groups:

A. Perfect analgesia and good relaxation, inability to raise the legs from the table or to contract the rectus abdominis. Operation conducted without supplements, comparable to subarachnoid spinal anaesthesia.

B. Satisfactory analgesia for surgery without supplements, but relaxation incomplete, tactile and pressure sensations permissible if not painful.

C. Satisfactory analgesia for surgery with intravenous pethidine 100 mg or thiopentone, administered after the incision was completed, for pain from deeper tissues. Pain due to recession of analgesia was excluded.

D. Analgesia not enough for incision without light narcosis, application of towel clips caused only discomfort.

E. Hypalgesia five segments or more, negligible to slight motor weakness and sympathetic block, towel clips painful, narcosis essential.

This classification only applied to surgery conducted within the anaesthetized zone and an extension of surgical stimuli, for example, to unanaesthetized upper abdomen or pelvis was discounted. A few cases with excellent relaxation equivalent of group A, but complaining of pain on incision, were classified as group B. Personal judgment was unavoidable in allowing for the more intense stimuli arising from visceral traction; cases classified as B for inguinal herniotomy

TABLE II
*Details of solutions tested.*

| Name of drug | Strength | Mean dose (mg) | Other constituents |
|---|---|---|---|
| Procaine hydrochloride B.P. | 2% | 610 | 0.9% sodium chloride |
| | 2% | 609 | 0.9% sodium chloride adrenaline |
| | 1% | 486 | 0.9% sodium chloride |
| | 1% | 462 | 0.9% sodium chloride adrenaline |
| Butethamine formate N.N.R. | 1% | 455 | 0.9% sodium chloride |
| | 1% | 441 | 0.9% sodium chloride adrenaline |
| Amethocaine hydrochloride B.P. | 1/300 | 88 | 0.9% sodium chloride |
| | 1/300 | 88 | 0.9% sodium chloride adrenaline |
| | 1/500 | 76 | 0.9% sodium chloride |
| | 1/500 | 77 | 0.9% sodium chloride adrenaline |
| | Viscous (preliminary saline) | 86 | 6% polyvinylpyrrolidone adrenaline 1/140,000 |
| | Viscous (terminal saline) | 89 | 6% polyvinylpyrrolidone adrenaline 1/140,000 |
| Tutocaine (p-amino-benzoyl dimethyl-amino-methyl butanol hydrochloride) | 1% | 387 | 0.9% sodium chloride |
| | 1% | 361 | 0.9% sodium chloride adrenaline |
| Amylocaine hydrochloride B.P.C. | 2% | 501 | 0.5% sodium chloride 2% glucose |
| | 2% | 548 | 0.5% sodium chloride 2% glucose adrenaline |
| Piperocaine hydrochloride U.S.P. | 2% | 490 | nil |
| | 2% | 514 | adrenaline |
| | 1% | 349 | 0.45% sodium chloride |
| | 1% | 353 | 0.45% sodium chloride adrenaline |
| Hexylcaine hydrochloride N.N.R. | 1% | 373 | nil |
| | 1% | 374 | adrenaline |
| Diethoxin hydrochloride (beta diethylaminoethyl-p-ethoxy benzoate hydrochloride) | 2% | 571 | nil |
| | 2% | 585 | adrenaline |
| | 1% | 415 | nil |
| | 1% | 405 | adrenaline |
| Lignocaine hydrochloride B.P.C. | 2% | 503 | nil |
| | 2% | 458 | adrenaline |
| | 1% | 316 | 0.45% sodium chloride |
| | 1% | 314 | 0.45% sodium chloride adrenaline |
| Carbocaine (d-l-N methyl-pipecolic acid 2-6-dimethylanilide) | 1% | 235 | adrenaline |

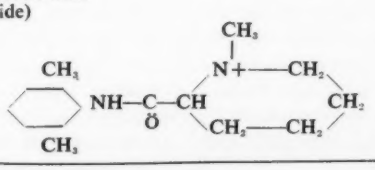
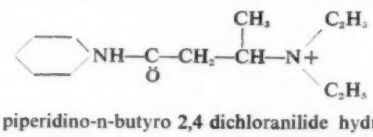


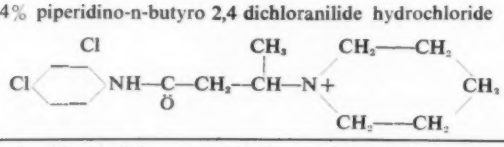
TABLE II—*continued*

| Name of drug | Strength | Mean dose (mg) | Other constituents |
|---|---|---|---|
| Amplicaine | 2% | 710 | nil |
| | 2% | 750 | adrenaline |

2% beta diethyl-amino-n-butyro-anilide hydrochloride



0.4% piperidino-n-butyro 2,4 dichloranilide hydrochloride



| | | | |
|---|---|---|---|
| Cinchocaine hydrochloride B.P. | 1/300 | 98 | 0.27% sodium chloride 3% glucose |
| | 1/300 | 90 | 0.27% sodium chloride 3% glucose adrenaline |
| | 1/600 | 58 | 0.45% sodium chloride |
| | 1/600 | 63 | 0.45% sodium chloride adrenaline |

or gastro-enterostomy might easily prove to be C for appendicectomy or gastrectomy respectively. Since the majority of cases were categorized on the basis of stimuli arising from the parietes, reclassification as a result of reaction to prolonged visceral traction was avoided in order to preserve uniformity of comparison as far as possible. Reclassification from B to C was avoided in cases where supplements were given only to provide the surgeon with greater relaxation as frequently occurred with Tutocaine. The distribution of cases according to this scheme is shown in table III.

A consideration of the unsupplemented cases for each solution gives an indication of relative potency (table IV). In the case of distribution which shows a sharp peak at a given intensity of block—for example, lignocaine, Tutocaine and procaine—it seems reasonable to assume that the sampling was adequate, but other solutions showing irregular distribution presumably require larger case samples in order to provide a satisfactory picture of average intensity of action. The less satisfactory samples have been marked with an asterisk in table IV and fortunately the powerful drugs have given good distribution curves.

Perhaps the most significant finding was the augmentation of the degree of nerve block produced by the addition of adrenaline. Whilst adrenaline is usually added to anaesthetic solutions to prolong anaesthesia and to limit systemic reactions, increased intensity of motor and sensory block was demonstrable with all the solutions except three. The mean intensity obtained was about the same for the solutions with and without adrenaline for 1 per cent hexylcaine, 1 per cent butethamine and 1 per cent procaine, but in spite of the absence of potentiation by adrenaline there was narrowing of the range of response and the anaesthesias became more uniform in intensity. Increasing the concentration of procaine to 2 per cent did bring about adrenaline potentiation and it is possible that different concentrations and greater numbers of cases would result in similar findings for hexylcaine and butethamine. One per cent lignocaine with adrenaline gave a better blockade than 2 per cent lignocaine plain, and similarly 1 per cent piperocaine with adrenaline was stronger than

TABLE III

*Case distribution according to intensity of nerve block, categories A to E described in text.*

| Solution | Without adrenaline | | | | | With adrenaline | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | A | B | C | D | E | A | B | C | D | E |
| Procaine 2% | 3 | 10 | 5 | 2 | – | 6 | 10 | 3 | 1 | – |
| Procaine 1% | – | 1 | 3 | 8 | 8 | – | 1 | 2 | 11 | 6 |
| Butethamine 1% | 2 | 4 | 4 | 7 | 3 | 1 | 5 | 5 | 8 | 1 |
| Amethocaine 1/300 | 5 | 6 | 5 | 4 | – | 13 | 5 | 1 | 1 | – |
| Amethocaine 1/500 | 1 | 4 | 3 | 11 | 1 | 6 | 3 | 4 | 7 | – |
| Amethocaine viscous preliminary saline | – | – | – | – | – | 9 | 4 | 5 | 2 | – |
| Amethocaine viscous terminal saline | – | – | – | – | – | 5 | 5 | 8 | 2 | – |
| Tutocaine 1% | – | 4 | 6 | 10 | – | – | 12 | 7 | 1 | – |
| Amylocaine 2% | – | 5 | 2 | 10 | 3 | 1 | 9 | 3 | 6 | 1 |
| Piperocaine 2% | 3 | 3 | 7 | 5 | 2 | 5 | 5 | 4 | 4 | 2 |
| Piperocaine 1% | – | 5 | 5 | 9 | 1 | 2 | 6 | 4 | 8 | – |
| Hexylcaine 1% | 2 | 9 | 3 | 5 | 1 | 2 | 7 | 8 | 2 | 1 |
| Diethoxin 2% | 4 | 5 | 3 | 6 | 2 | 6 | 10 | 2 | 1 | 1 |
| Diethoxin 1% | – | 4 | 6 | 9 | 1 | – | 9 | 8 | 3 | – |
| Lignocaine 2% | 7 | 9 | 3 | 1 | – | 12 | 7 | 1 | – | – |
| Lignocaine 1% | 1 | 7 | 5 | 4 | 2 | 10 | 5 | 4 | 1 | – |
| Carbocaine 1% | – | – | – | – | – | 3 | 8 | 5 | 3 | 1 |
| Amplicaine 2% | – | 4 | 5 | 6 | 5 | 1 | 6 | 5 | 6 | 2 |
| Cinchocaine 1/300 | – | 2 | 4 | 12 | 2 | 4 | 3 | 5 | 6 | 2 |
| Cinchocaine 1/600 | – | 1 | 4 | 7 | 8 | – | 2 | 7 | 7 | 4 |

2 per cent piperocaine plain. The number of unsupplemented anaesthesias was trebled by the addition of adrenaline to 1 per cent Tutocaine and to 1/300 cinchocaine, and doubled for 2 per cent amylocaine and 1 per cent diethoxin.

In table V are incidental findings which may be of interest although they are only approximations. The A/B ratio gives an indication of motor block. In addition, the category of muscle relaxation was noted in all cases as good, moderate, slight or nil and the ratio $\frac{\text{Good}+\text{Moderate}}{\text{Slight}+\text{Nil}}$ has been obtained for all the weaker solutions where the majority of cases were in categories C to E. From the two ratios drugs have been grouped in order of relative intensity of motor blocking activity in table V. Dilatation of veins of legs, warming of toes and congestion of the penis to an obvious degree was evidence of sympathetic blockade and drugs have been similarly arranged in order of activity. Although lignocaine gave a 50 per cent development of anaesthesia much sooner than other drugs, the time for full onset or about 90 per cent spread did not differ markedly. Drugs have been arranged in order of speed of full onset of anaesthesia in table V and details are given in table VI. Evaluation of duration of anaesthesia can only be made accurately when the operation outlasts the paralysis and analgesia and when the patient is conscious. The recession of analgesia and of paresis was noted every time it occurred during operation in the unsupplemented anaesthesias and the time of recession is taken from full onset (table VI). Motor recession preceded pain by some 10 to 30 minutes with lignocaine, carbocaine and amplicaine, and general anaesthesia had to be undertaken to provide relaxation in quite a number of cases. The longest time at which painless skin closure took place when greater than the mean recession time has also been noted; naturally the number of cases depended on an uncontrollable variable, the duration of surgery. The estimated probable average duration is based on the figures in the two preceding columns of table VI and upon postoperative observations of skin analgesia, admittedly a less satisfactory method. Lignocaine, diethoxin and hexylcaine appear to be of about equal duration. The fall in blood pressure was looked for in every case with the object of comparing the pressor blocking activity of different drugs and it was found that the number of cases with severe falls of blood pressure corresponded fairly closely to the number of high

TABLE IV

*Relative potency of test solutions (for sensory block).*

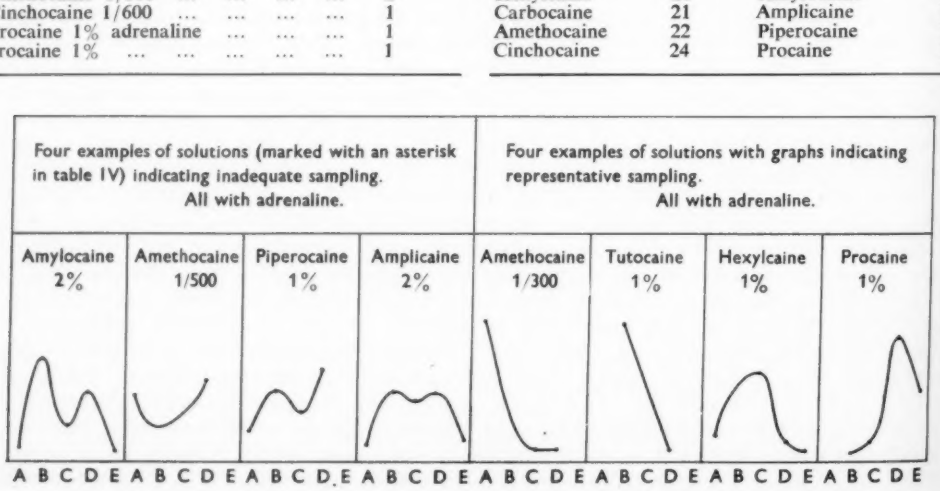
| Solution in order of apparent potency | Number of unsupplemented anaesthesias |
|---|---|
| Lignocaine 2% adrenaline ... ... ... | 19 |
| Amethocaine 1/300 adrenaline ... ... | 18 |
| Lignocaine 2% ... ... ... ... ... | 16 |
| Procaine 2% adrenaline ... ... ... | 16 |
| Diethoxin 2% adrenaline ... ... ... | 16 |
| Lignocaine 1% adrenaline ... ... ... | 15 |
| Procaine 2% ... ... ... ... ... | 13 |
| Tutocaine 1% adrenaline ... ... ... | 12 |
| Amethocaine 1/300 ... ... ... ... | 11 |
| Carbocaine 1% adrenaline ... ... ... | 11 |
| Hexylcaine 1% ... ... ... ... ... | 11* |
| Piperocaine 2% adrenaline ... ... | 10* |
| Amylocaine 2% adrenaline ... ... | 10* |
| Amethocaine 1/500 adrenaline ... ... | 9* |
| Diethoxin 2% ... ... ... ... ... | 9* |
| Hexylcaine 1% adrenaline ... ... ... | 9 |
| Diethoxin 1% adrenaline ... ... ... | 9 |
| Piperocaine 1% adrenaline ... ... | 8* |
| Lignocaine 1% ... ... ... ... ... | 8 |
| Cinchocaine 1/300 adrenaline ... ... | 7* |
| Amplicaine 2% adrenaline ... ... | 7* |
| Piperocaine 2% ... ... ... ... | 6* |
| Butethamine 1% adrenaline ... ... | 6 |
| Butethamine 1% ... ... ... ... | 6 |
| Amethocaine 1/500 ... ... ... ... | 5* |
| Piperocaine 1% ... ... ... ... | 5* |
| Amylocaine 2% ... ... ... ... | 5* |
| Tutocaine 1% ... ... ... ... ... | 4 |
| Diethoxin 1% ... ... ... ... ... | 4 |
| Amplicaine 2% ... ... ... ... | 4 |
| Cinchocaine 1/600 adrenaline ... ... | 2 |
| Cinchocaine 1/300 ... ... ... ... | 2 |
| Cinchocaine 1/600 ... ... ... ... | 1 |
| Procaine 1% adrenaline ... ... ... | 1 |
| Procaine 1% ... ... ... ... ... | 1 |

TABLE V

*Drugs in approximate order of intensity of action of motor and sympathetic block, and onset and duration of anaesthesia.*

| Relative intensity of motor to sensory block | | Intensity of sympathetic block | |
|---|---|---|---|
| Amethocaine | | Lignocaine, excellent | |
| Lignocaine, Cinchocaine, Diethoxin | similar | Amethocaine, Cinchocaine, Tutocaine, Butethamine | good |
| Piperocaine | | Amylocaine, moderate | |
| Carbocaine, Hexylcaine, Procaine | similar | Piperocaine, variable early (10) | |
| | | Procaine, variable late (20) | |
| Butethamine, Amylocaine, Amplicaine | similar | Others variable | |
| Tutocaine | | | |

| Speed of onset | min | Duration of anaesthesia | min |
|---|---|---|---|
| Tutocaine | 14 | Cinchocaine | 210 |
| Diethoxin | 16 | Amethocaine | 170 |
| Lignocaine | 16 | Lignocaine | 100 |
| Procaine | 16 | Diethoxin | 100 |
| Amylocaine | 17 | Hexylcaine | 90 |
| Piperocaine | 17 | Tutocaine | 80 |
| Butethamine | 17 | Carbocaine | 80 |
| Amplicaine | 18 | Butethamine | 80 |
| Hexylcaine | 20 | Amylocaine | 80 |
| Carbocaine | 21 | Amplicaine | 70 |
| Amethocaine | 22 | Piperocaine | 70 |
| Cinchocaine | 24 | Procaine | 55 |


Four examples of solutions (marked with an asterisk in table IV) indicating inadequate sampling. All with adrenaline.
Four examples of solutions with graphs indicating representative sampling. All with adrenaline.
Amylocaine 2%
Amethocaine 1/500
Piperocaine 1%
Amplicaine 2%
Amethocaine 1/300
Tutocaine 1%
Hexylcaine 1%
Procaine 1%
A B C D E A B C D E A B C D E A B C D E A B C D E A B C D E A B C D E A B C D E A B C D E

blocks in the series, as might be expected. Although procaine appeared to give more cases of severe hypotension than the others, very large numbers of strictly comparable cases would be required to assess any difference in vasomotor blockade.

### TOXICITY

After intensity of action the most important consideration is toxicity and quite a few of the formidable number of toxic effects listed by Moore (1956) were encountered in these patients. They have been presented in detail and include twelve cases from the trial series mentioned in the introduction and one case of convulsions. Broadly the reactions appear to be of two types, those occurring during or in the first few minutes after injection and those appearing after 10 minutes. Early reactions comprised sweating, vomiting, transient fall of blood pressure, shivering, rigidity of limbs, vertigo, grunting, shouting, delirium, jactitation, transient loss of consciousness, and convulsions. The case of convulsions was a thin patient aged 60, with 18 months history of prostatic obstruction, Hb 70 per cent, blood urea 45 mg per cent, and blood pressure 125/80 mm Hg. Twenty ml of 2 per cent diethoxin were injected in the third lumbar space with

TABLE VI

*Data on onset and duration of anaesthesia in minutes from which the figures in table V have been obtained.*

| Solution | Mean time of full onset | Range of onset | Cases of recession, time from full onset m=motor; s=sensory | Longest time for painless closure | Probable duration |
|---|---|---|---|---|---|
| Procaine 2% ... ... ... | 13 | 10–16 | s. 30, 35, 40 | 70 | 60 |
| Procaine 2% adrenaline ... | 15 | 10–18 | s. 65, 70 | | 75 |
| Procaine 1% ... ... ... | 16 | 10–20 | | | 45 |
| Procaine 1% adrenaline ... | 19 | 15–22 | s. 50, 80 | | 65 |
| Butethamine 1% ... ... | 18 | 13–30 | s. 55, 80 | 75 | 70 |
| Butethamine 1% adrenaline | 17 | 5–25 | s. 60, 75, 90 | 85 | 90 |
| Amethocaine 1/300 ... ... | 22 | 10–30 | | 180 | >180 |
| Amethocaine 1/300 adrenaline | 26 | 20–30 | | 110 | >200 |
| Amethocaine 1/500 ... ... | 17 | 10–25 | | | 120 |
| Amethocaine 1/500 adrenaline | 24 | 17–37 | s. 105, 135 | 140 | 130 |
| Amethocaine viscous adrenaline | 26 | 15–40 | | 180 | >200 |
| Tutocaine 1% ... ... ... | 13 | 10–18 | s. 50 | 75 | 70 |
| Tutocaine 1% adrenaline ... | 16 | 12–25 | s. 80, 95 | 85 | 90 |
| Amylocaine 2% ... ... | 16 | 10–25 | s. 50, 68 | 75 | 70 |
| Amylocaine 2% adrenaline | 19 | 10–30 | s. 60, 65 | 100 | 90 |
| Piperocaine 2% ... ... | 15 | 10–20 | s. 50, 50, 60 | 60 | 60 |
| Piperocaine 2% adrenaline ... | 18 | 10–25 | s. 70 | 70 | 85 |
| Piperocaine 1% ... ... | 17 | 14–22 | s. 55 | | 70 |
| Piperocaine 1% adrenaline ... | 17 | 12–27 | s. 50, 55, 75, 80, 80 | | 75 |
| Hexylcaine 1% ... ... | 20 | 15–30 | s. 40, 95 | 80 | 70 |
| Hexylcaine 1% adrenaline ... | 20 | 15–35 | s. 50, 50, 55, 70 | 160 | 115 |
| Diethoxin 2% ... ... ... | 17 | 12–30 | s. 80 | | 100 |
| Diethoxin 2% adrenaline ... | 14 | 8–25 | s. 110 | 125 | 130 |
| Diethoxin 1% ... ... ... | 16 | 10–20 | s. 60, 70, 70 | 85 | 80 |
| Diethoxin 1% adrenaline ... | 18 | 10–24 | s. 65, 70, 100 | 95 | 95 |
| Lignocaine 2% ... ... | 16 | 5–25 | s. 65, 75, 75, 100; m. 50, 60, 60, 65 | | 95 |
| Lignocaine 2% adrenaline ... | 20 | 12–28 | s. 80, 100, 110. m. 60 | 110 | 110 |
| Lignocaine 1% ... ... | 14 | 8–20 | s 80, 90; m. 40, 85. | | 80 |
| Lignocaine 1% adrenaline ... | 16 | 10–25 | | 90 | 110 |
| Carbocaine 1% adrenaline ... | 21 | 12–25 | s. 65, 70, 85; m. 40, 60 | | 80 |
| Amplicaine 2% ... ... | 18 | 12–25 | s. 60, 65; m. 45 | | 65 |
| Amplicaine 2% adrenaline ... | 19 | 12–25 | s. 55, 60; m. 75 | 80 | 80 |
| Cinchocaine 1/300 ... ... | 22 | 15–30 | | 155 | >200 |
| Cinchocaine 1/300 adrenaline | 27 | 15–50 | | | >250 |
| Cinchocaine 1/600 ... ... | 23 | 15–35 | s. 160 | | 180 |
| Cinchocaine 1/600 adrenaline | 22 | 15–30 | | 205 | 220 |

moderate difficulty, aspiration tests being negative. During the customary pause following the first 10 ml of solution the patient began shouting and shivering. This subsided and the second 20 ml were injected. Immediately following this he became confused, recommenced shouting incoherently and within one minute he started to convulse. The first fit lasted 8 seconds, followed by relaxation and coma with deep respirations. Fits recurred at about 10-second intervals and he had six clonic convulsions before control was obtained with 250 mg of 2½ per cent thiopentone. Respiration was present throughout. Consciousness had nearly returned 20 minutes after the onset and no analgesia was demonstrable. General anaesthesia was given for the prostatectomy and recovery was uneventful. The case quoted by Bromage (1954b) was similar and he considers rapid absorption from the extradural space was responsible. Absorption giving rise to subconvulsive symptoms detailed in table VII indicates that this phenomenon appears often, and although anatomical differences may account for rapid absorption it seems that certain drugs are more dangerous than others. Whilst sensitivity to premedicating drugs cannot be excluded, experience with intrathecal spinal anaesthesia in similar patients indicates that it is uncommon and mainly confined to vomiting. Signs of early toxicity were most marked with diethoxin, amylocaine, Amplicaine, piperocaine and procaine; hypotension occurred with procaine, Amplicaine, amylocaine and piperocaine.

Lignocaine figures prominently in delayed reactions attributable to late absorption. It is impossible to say how close the blood level for coma approaches the convulsive level but this late absorption cannot be regarded as devoid of danger. Nevertheless since it is slower in onset, adequate precautions may be taken and generalized analgesia is not unhelpful in surgery. The control of toxic symptoms from procaine has been well established in intravenous therapeutic analgesia and the usual emergency facilities; 2½ per cent thiopentone, oxygen, apparatus for intubation, suction and 1/200,000 noradrenaline should be immediately available. The lowest incidences of all types of toxic reaction were found with amethocaine, Carbocaine and cinchocaine. Adrenaline tended to protect against toxic reactions but the total incidence of such reactions was not reduced by halving the concentration of lignocaine, diethoxin and procaine.

### OTHER FINDINGS

In table VIII it has been shown that with increasing age a greater intensity of nerve block results from all the solutions used in these cases, considered collectively.

Bromage (1954c) noted that there was a strong association between age and dosage, less of an anaesthetic solution being required for an equivalent degree of block as age progresses, this being attributed to anatomical changes. It would be difficult to say to what extent the apparently greater spread is due to enhanced nerve penetration or nerve susceptibility, since, as the block becomes more intense so the spread appears greater, that is, hypalgesic zones become analgesic. In fact this may be the explanation for the difference noticed by Bromage (1954d) in the spread of equal quantities of 1/600 cinchocaine and 1.2 per cent lignocaine, the latter giving the more extensive anaesthesia, as it is much more powerful.

Whilst the negative pressure of the extradural space is known to vary, so also did the injection characteristics of these patients. The same needles were used in the 740 cases and ease or difficulty of injection was recorded at the time. Twelve per cent were difficult, considerable force being required and 7 per cent were easy, hardly any pressure being needed up to the end of the injection. Neither ease nor difficulty of injection was particularly associated with good or poor anaesthesia with flow back or with toxic symptoms. In 9 per cent of cases there was marked flow back into the syringe, suggestive of thecal puncture. Drip back was usual in most cases but the pressure and rate fell off rapidly. Marked drip back was usual in cases of reflux on aspiration. In one extreme case the whole of the anaesthetic solution (40 ml) could be aspirated into the syringe, as is occasionally possible when the needle point is incorrectly placed in ligament, but proof was obtained of correct entry. Great resistance with pain on injection was sometimes encountered in cases of prolapsed disc, presumably from adhesions. Because of the

TABLE VII

*Systemic toxic reactions.*

Numbers ..... Time of onset from injection in minutes.
Numbers in brackets ..... Duration of symptom in minutes.
Coma ..... Not awakened by painful stimuli.
Comatose ..... Transient awakening with pain.
Dizzy ..... Distressing vertigo.

| Solution | | Symptoms | Dose (mg) |
|---|---|---|---|
| Procaine 2% | 5 | Semiconscious, pulseless, pale | 500 |
| | 10 | Comatose | 680 |
| | 12 | Generalized analgesia | 600 |
| | 23 | Drowsy | |
| Procaine 2% adrenaline | 40 | Comatose | 640 |
| Procaine 1% | 0 | Shivering | 400 |
| | 3 | Dizzy, vomiting, hypotension | 500 |
| | 7 | Pale, vomiting, pulseless | 500 |
| Procaine 1% adrenaline | 1 | Shivering, dizzy | 370 |
| | 2 | Dizzy | 500 |
| | 2 | Dizzy, confused | 440 |
| | 3 | Confused, semiconscious, 15 coma (180) | 400 |
| | 5 | Dizzy | 500 |
| | 20 | Drowsy, generalized analgesia | 500 |
| Butethamine 1% | 3 | Drowsy (25) | 500 |
| | 5 | Dizzy (5) Generalized analgesia, drowsy | 500 |
| | 5 | Dizzy (10) | 450 |
| | 7 | Drowsy | 500 |
| | 7 | Comatose, cyanotic (20) generalized analgesia | 450 |
| | 8 | Comatose | 350 |
| | 10 | Drowsy, 20, comatose | 500 |
| | 15 | Drowsy | 500 |
| Butethamine 1% adrenaline | 3 | Dizzy, 10 drowsy | 460 |
| | 3 | Vomiting, 22 cyanotic drowsy (60) | 450 |
| | 4 | Nausea, pallor, drowsiness, 10 comatose cyanotic (20) | 400 |
| | 10 | Cyanotic, drowsy | 400 |
| | 14 | Drowsy | 300 |
| | 15 | Drowsy | 460 |
| | 20 | Drowsy | 450 |
| Amethocaine 1/300 | | Nil | |
| Amethocaine 1/300 adrenaline | | Nil | |
| Amethocaine 1/500 | 15 | Shivering | 80 |
| Amethocaine 1/500 adrenaline | | Nil | |
| Amethocaine viscous | 5 | Dizzy | 90 |
| | 25 | Confused, delirious | 90 |
| Tutocaine 1% | 5 | Dizzy, sweating | 400 |
| | 6 | Dizzy, nausea | 400 |
| Tutocaine 1% adrenaline | 5 | Shivering, jactitation of legs | 300 |
| Amylocaine 2% | 1 | Sweating, transient unconsciousness, jactitation, hypotension, pallor | 600 |
| | 2 | Confused, restless, vomiting, sweating, hypotension | 500 |
| | 15 | Drowsy, pale, generalized analgesia | 440 |

TABLE VII *continued*

| Solution | | Symptoms | Dose (mg) |
|---|---|---|---|
| Amylocaine 2% adrenaline | 1 | Vomiting | 400 |
| | 2 | Shivering | 600 |
| | 20 | Generalized analgesia | 540 |
| | 20 | Confused, comatose | 600 |
| Piperocaine 2% | 4 | Sweating, 20 generalized analgesia | 500 |
| | 17 | Nausea, drowsy | 400 |
| | 20 | Generalized analgesia | 520 |
| | 20 | Comatose | 500 |
| Piperocaine 2% adrenaline | | Nil | |
| Piperocaine 1% | 7 | Confused, rigid, pale, pulseless, transient unconsciousness | 400 |
| Piperocaine 1% adrenaline | 3 | Shivering | 340 |
| | 6 | Sweating | 400 |
| Hexylcaine 1% | | Nil | |
| Hexylcaine 1% adrenaline | 0 | Twitching of limbs, drowsy | 370 |
| | 0 | Shivering | 400 |
| | 1 | Dizzy, hypotension, 15 generalized analgesia drowsy | 370 |
| | 3 | Dysarthric, comatose, 10 shivering | 390 |
| Diethoxin 2% | 0 | Confusion, shouting, delirium, convulsion | 400 |
| | 13 | Transient unconsciousness, rigid, cyanotic (3), confused | 720 |
| Diethoxin 2% adrenaline | 12 | Nausea, 27 pallor, coma (75) | 580 |
| Diethoxin 1% | 0 | Shivering | 500 |
| | 0 | Dizzy, comatose | 400 |
| | 1 | Shivering | 400 |
| | 2 | Dizzy | 450 |
| Diethoxin 1% adrenaline | 0 | Shivering | 400 |
| | 1 | Shivering | 450 |
| | 2 | Shivering, dizzy, tachycardia, 10 comatose, delirious, dysarthric, cyanotic, shallow respiration | 350 |
| | 4 | Vomiting | 400 |
| | 4 | Drowsy | 350 |
| | 7 | Dizzy | 400 |
| Lignocaine 2% | 8 | Drowsy | 500 |
| | 10 | Drowsy, dysarthric, 25 coma (60) | 480 |
| | 12 | Generalized analgesia, 30 coma (40) | 800 |
| | 20 | Generalized analgesia, comatose | 600 |
| | 35 | Drowsy | 540 |
| | 40 | Coma (30) | 500 |
| Lignocaine 2% adrenaline | 30 | Drowsy | 500 |
| | 40 | Coma (120) | 580 |
| | 40 | Shallow respiration, cyanotic, 55 comatose | 400 |
| | 45 | Drowsy, shallow respiration, cyanotic | 320 |
| Lignocaine 1% | 8 | Drowsy | 370 |
| | 10 | Generalized analgesia, coma (80), respiration 14/min. | 310 |
| | 14 | Comatose, confused, twitching fingers | 300 |
| | 16 | Generalized analgesia | 400 |
| | 20 | Comatose | 330 |
| | 100 | Drowsy, twitching of fingers | 320 |

TABLE VII—*continued*

| Solution | | Symptoms | Dose (mg) |
|---|---|---|---|
| Lignocaine 1% adrenaline | 15 | Drowsy, twitching of hand | 360 |
| | 15 | Pallor, coma (60) | 400 |
| | 20 | Drowsy | 380 |
| | 30 | Drowsy | 400 |
| Carbocaine 1% adrenaline | 4 | Dizzy | 200 |
| Amplicaine 2% | 3 | Dizzy | 800 |
| | 5 | Drowsy | 800 |
| | 5 | Dizzy | 800 |
| | 6 | Jactitation, grunting, transient loss of consciousness, pallor, hypotension, 20 confusion, dysarthria, tremor | 800 |
| | 7 | Dizzy, 15 drowsy | 700 |
| | 10 | Twitching of limbs | 800 |
| | 10 | Confusion, dysarthria, drowsiness, hypotension, vertigo, nystagmus, twitching (20) | 800 |
| | 20 | Dizzy (30) | 500 |
| Amplicaine 2% adrenaline | 0 | Shivering, twitching, dizzy | 600 |
| | 6 | Nausea, retching | 800 |
| | 15 | Drowsy (100) | 800 |
| | 15 | Sweating, vomiting, hypotension | 800 |
| | 20 | Tremor, drowsiness | 700 |
| | 25 | Comatose | 600 |
| Cinchocaine 1/300 | | Nil | |
| Cinchocaine 1/300 adrenaline | | Nil | |
| Cinchocaine 1/600 | 0 | Transient confusion, grunting, twitching | 33 |
| Cinchocaine 1/600 adrenaline | 0 | Transient unconsciousness (4), twitching, rigidity | 67 |

possibility of rapid systemic absorption with toxic manifestations being due to damage to small vessels, the incidence of bloodstained reflux was of interest. Nine cases (1.2 per cent) showed reflux of small quantities of bloodstained fluid, none of them was associated with toxic symptoms. In 13 cases, thecal tap was performed accidentally and the anaesthetic solution was subsequently injected either at the same or at an adjacent space without clinical evidence of entry of the drug into the theca. Once subarachnoid anaesthesia did occur.

TABLE VIII

| Age group | Number of cases | Per cent unsupplemented anaesthesias (A+B) |
|---|---|---|
| Up to 20 | 81 | 32 |
| 21 to 30 | 171 | 33 |
| 31 to 40 | 174 | 43 |
| 41 to 50 | 135 | 49 |
| 51 to 60 | 121 | 50 |
| 61 + | 58 | 54 |

The patient, aged 40, was a well nourished fellah with bilateral ureterolithiasis secondary to bilharziasis. One per cent procaine in saline was injected in the third lumbar space, he complained of feeling dizzy, and unco-operative struggling occurred after 20 ml and a fluid jet under great pressure came from the needle. The needle was withdrawn and the patient placed supine. No analgesia developed in 10 minutes and a further 30 ml of procaine were introduced in the same space without drip back or flow back. In 15 minutes there was complete paralysis and anaesthesia to the second thoracic segment which lasted two hours. Since 1 per cent procaine does not give such a complete anaesthesia extradurally, partial entry into the theca is presumed.

Most authorities, for example Dogliotti (1939) and Pitkin (1946), rely on choice of interspace for distribution of the anaesthetic solution, but Bromage (1954e) mentions the use of a head down tilt for cephalic spread and a sitting posi-

tion to limit the upward spread. It was decided to investigate the precise extent of gravitational spread to the lumbosacral region. For this purpose cases were classified into two groups as regards site of injection and into five groups as regards downward spread. These data were abstracted from 663 cases as the other postural techniques were too few to warrant inclusion and cases of saline filling were excluded. The results are shown in table IX. The comparative intensity of block was more difficult to determine as the critical test of tolerance to intrapelvic or perineal surgery was only applicable in a limited number of conscious cases. However, it is noted that a maximum of 14 per cent were classified "S5 good". Although it would be desirable to know what is the exact expectation of sacral analgesia, equally intense as that of the lower thoracic region, obtained by optimal gravitational movement of solution, this can only be estimated as certainly not greater than 30 per cent. From "% S5" the overall influence of gravity is definite and the maintenance of the sitting position for at least 5 minutes after lumbar injection gave approximately a two-thirds chance of obtaining complete sacral hypalgesia.

By the use of the lateral position predominantly unilateral analgesia is obtained in a large proportion of cases, but quite often it was found that more intense analgesia resulted on the upper side. It is assumed that this paradoxical block is due to puncture of the extradural space to the wrong side of the midline. Since completing this investigation the author has used the Tuohy needle to provide a directional as well as a gravitational movement of anaesthetic solutions and the impression has been gained that, for maximal lumbosacral or thoracocervical distribution of the anaesthetic, the injection should be made at L4 interspace, bevel down in the sitting position or at T12 or L1 interspace bevel up (cephalic), in a steep head down lateral position, respectively. In each case the position must be maintained for at least 5 minutes.

TABLE IX

*Lumbosacral penetration in relation to postural technique.*

Above L3 .....Hypalgesia not reaching the front of the knee.
L3 .....Hypalgesia reaching front of knee.
L5 .....Hypalgesia to front of leg.
S2 .....Hypalgesia to back of knee.
S5 .....Hypalgesia to anal margin.
S5 poor .....Intrapelvic surgery painful.
S5 patchy .....Some roots escaped analgesia.
S5 good .....Intrapelvic surgery painless.
% S5 .....% of one postural technique with hypalgesia to anal margin.

| Position | Case distribution for hypalgesia or analgesia | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Above L3 | L3 | L5 | S2 | S5 | S5 poor and S5 patchy | S5 good | %S5 |
| Injection sitting 5 minutes sitting Puncture L1 to L4 | 7 | 13 | 34 | 28 | 148 | 38 (26%) | 21 (14%) | 64 |
| Injection sitting 5 minutes lateral or supine Puncture L1 to L4 | 10 | 9 | 20 | 16 | 77 | 16 (21%) | 5 (6%) | 58 |
| Injection lateral 5 minutes lateral or supine Puncture L1 to L4 | 10 | 30 | 42 | 39 | 87 | — | — | 42 |
| Injection lateral 5 minutes lateral or supine Puncture T12 or higher | 30 | 10 | 29 | 7 | 17 | — | — | 18 |

TABLE X
*Volume-segment distribution of 660 cases.*

| Volume (ml) | Segments of hypalgesia | | | | | Mean segmental block |
|---|---|---|---|---|---|---|
| | 5–9 | 10–14 | 15–19 | 20–24 | 25–30 | |
| 45 and greater | 1 | 6 | 39 | 4 | – | 16 |
| 40–44 | 3 | 46 | 83 | 20 | 2 | 17 |
| 35–39 | 2 | 30 | 41 | 18 | 3 | 16 |
| 30–34 | 20 | 46 | 79 | 14 | – | 14 |
| 25–29 | 18 | 47 | 44 | 7 | 3 | 14 |
| 24 and less | 16 | 32 | 30 | 5 | 1 | 14 |

TABLE XI
*Volume-segment relationship of 200 cases of "B" intensity of nerve block.*

| Volume (ml) | Range of segmental block | Mean segmental block | No. of cases |
|---|---|---|---|
| 45 & greater | 16–23 | 19.8 | 13 |
| 40–44 | 12–23 | 17.6 | 47 |
| 35–39 | 10–26 | 15.1 | 26 |
| 30–34 | 6–24 | 15.4 | 51 |
| 25–29 | 6–20 | 13.3 | 38 |
| 24 & less | 8–20 | 14.4 | 25 |

Since the descent of anaesthetic solution is variable, it is suggested that studies of the relationship between volume and segmental block might incorporate data of lumbosacral penetration as well as the extent of cephalic spread. In table X this relationship only shows a dubious general tendency for a larger volume to give a more extensive block. The fact that potency influences apparent spread has already been suggested and owing to the great range of nerve blocking activity of the different solutions, any simple relationship between volume and extent of spread might be concealed. If that were the case the potency factor could be eliminated by examining the volume-segment relationship in one category of nerve block. Category "B" was probably the most accurately delineated type of block and in table XI it is obvious that the relationship has become only a little more definite. The enormous variation makes it virtually impossible to predict the total area of blockade produced by a given volume of solution.

Employing the principle of the injection of a viscous solution to limit the extent of block as practised by German workers, Bromage (1954f), using lignocaine 1.4 per cent with adrenaline and 7.5 per cent polyvinyl pyrrolidone, found that the ultimate spread did not appear to be limited in extent. The technique of Buchholz and Lesse (1950) consists of preliminary saline filling of the extradural space until resistance to injection occurs or pressure symptoms arise, then injection of 10 ml of solution containing 6 per cent polyvinyl pyrrolidone, adrenaline 1/150,000 and amethocaine hydrochloride in a dose ranging from 30 to 120 mg according to age and type of operation. Site of puncture, from the interspace of T2 to L3 also depends on the surgical requirements. The preliminary filling with saline is intended to open up the extradural space into the intervertebral foramina and to supply a hydrostatic cushion limiting the vertical spread of the anaesthetic solution. If it is true that limitation of spread is obtained in this way, then reversing the procedure by injecting the saline after the amethocaine should cause a very much wider spread. Accordingly two series of cases were conducted. In the first, the technique of Buchholz and Lesse was followed, except that the dosage of viscous solution was kept within a narrow range; in the second, the saline filling was performed last.

The simple 1-300 amethocaine hydrochloride in normal saline with 1/200,000 adrenaline forms a control series both for segmental block and for intensity of resultant anaesthesia. As all these amethocaine solutions gave good anaesthesia, segmental block was easy to determine in the manner already described. There was no case of recession and the longest time for painless skin closure was 180, 90 and 110 minutes for preliminary saline, terminal saline and 1/300 solution, respectively. Relaxation was generally excellent for all three. The results in table XII show surprisingly little difference in the distri-

TABLE XII

*Two techniques with viscous solutions and 1/300 amethocaine as control.*

| Solution | Mean time of full onset (min) | Mean number of segments and range | Number of unsupplemented anaesthesias | Mean dose (mg) and range | Mean volume of saline and range |
|---|---|---|---|---|---|
| Viscous amethocaine preliminary saline | 28<br>20–40 | 15<br>9–23 | 13 | 86<br>60–90 | 78<br>20–120 |
| Viscous amethocaine terminal saline | 24<br>15–35 | 17<br>11–26 | 10 | 89<br>75–105 | 90<br>52–180 |
| 1/300 amethocaine with adrenaline | 26<br>20–30 | 17<br>8–28 | 18 | 88<br>67–100 | — |

bution and degree of segmental block produced by the three techniques. There is some limitation of spread by the viscous solution preceded by saline filling and the range is less, but it is disappointing in degree. The 1/300 solution in equivalent dosage gave better results as regards intensity of block, but it would be improper to assume that this finding would be upheld by an extensive series of cases, particularly if conducted by an operator thoroughly experienced in the rather complicated German technique. Nevertheless the 1/300 solution is unlikely to give a less efficient block than the viscous solution and the extent of spread is only a little greater. It should be mentioned that the saline filling was rather a prolonged and generally unpleasant experience for the patient. In conclusion the author has the impression that the success of the German workers may not be so much due to their technique as to the fact that amethocaine is one of the two most powerful drugs available, has fewer toxic manifestations than lignocaine in equipotent doses, and gives longer anaesthesia than all the other local anaesthetics with the exception of cinchocaine. The use of doses which can be limited to 100 mg with adrenaline gives excellent and prolonged epidural anaesthesia. It is unfortunate that the drug has gained an unfavourable reputation because of absorption of toxic doses from mucous membranes in topical anaesthesia (Hewer, 1953).

It is perhaps unusual to consider the economic factor in therapeutics, but there is a tangible price difference in these drugs and prices have been calculated net per patient for purposes of comparison in table XIII. Only official names have been used except for the two preparations not yet listed, namely Tutocaine and Amplicaine.

TABLE XIII

*Comparative prices of drugs.*

| Drug | Sterling cost per patient | | Cheapest source |
|---|---|---|---|
| | *s.* | *d.* | |
| Procaine | 2 | 0 | British |
| Butethamine | 2 | 5 | American |
| Tutocaine | 2 | 6 | German |
| Diethoxin | 2 | 7 | American |
| Piperocaine | 2 | 8 | British |
| Amylocaine | 2 | 8 | British |
| Amethocaine | 2 | 9 | British |
| Amplicaine | 4 | 3 | Swiss |
| Cinchocaine | 5 | 0 | British |
| Lignocaine | 5 | 2 | French |
| Hexylcaine | 7 | 8 | American |

## MORBIDITY

Of fourteen postoperative deaths there was one which may be attributable to the anaesthetic, there were also one reactionary haemorrhage and one case of sepsis. Brief case notes are supplied.

(1) Thin poor specimen aged 60; operation one stage retropubic prostatectomy. Forty ml 1/500 amethocaine hydrochloride with adrenaline 1/200,000 at L4 space. Hypalgesia from C6 to S5, moderate but adequate relaxation of abdomen, sympathetic block and absent sweating over whole body. At 25 minutes the B.P. fell from 110/80 to 60/50 mm Hg, poor response to methylamphetamine, nearly avascular operating field. End of operation B.P. 70/40 mm Hg. Postoperatively 5 hours 110/80 mm Hg. Eight hours after operation hyperpyrexia 105°F, absent sweating, but urine output 50 ml. On the second day the patient developed anuria; epidural block (16 ml 1/300 amethocaine) resulted in 100 ml urine. On the 7th day anuria recurred and was unrelieved. Death on the 10th postoperative day, postmortem refused. It would

appear that hypotension caused an anoxaemic nephrosis in already damaged kidneys.

(2) Tall fit man aged 50 with 6 months history of sciatica. Operation laminectomy and removal of prolapsed disc. Forty ml of 1 per cent piperocaine with adrenaline 1/150,000 epidural. Hypalgesia T3 to L3, satisfactory for operation until sacral roots stimulated and supplements given. In 30 minutes B.P. fell from 110/90 to 70/50 mm Hg. Operating field nearly bloodless from combined local vasoconstriction and hypotension. Good progress until secondary haemorrhage (250 ml) from the wound on the 2nd day. Uneventful recovery.

(3) Well nourished man aged 20 with recurrent inguinal hernia. Twenty-five ml diethoxin 2 per cent with adrenaline at T12 interspace, 1½ inch (3 cm) deep, injection under considerable pressure and painful, no flow back. Hypalgesia T9 to L4, slight paresis right leg, operating conditions satisfactory, no supplements. Patient complained of pain in the back from time of operation but discharged on 7th day. Tenderness, swelling and redness over T12 to L2 spines was treated by private doctor with antibiotics. Readmitted to hospital in 5th week with hard red induration extending 3 inches either side of spine; aspiration gave a little pus which was sterile. Laseague negative and no apparent bony involvement.

Radiography gave a picture of marked osteoarthritis with osteophyte formation. Blood leucocytes 12,000. Induration persisted for another two months. Condition resolved by the 4th month after operation. The organism could not be identified and therefore the source of the cellulitis is a matter of speculation.

## DISCUSSION

Whilst the arrangement of drugs in order of potency in table IV cannot be taken as accurate in detail, the investigation does warrant an arrangement of drugs in broad order of potency of sensory blockade on the lines mentioned in the introduction. Roughly equivalent drugs are grouped together and in brackets are two which could not be tested in 2 per cent concentration. If both sensory and motor blockade were considered, the arrangement would be much the same except that Tutocaine would come after piperocaine.

$$\text{Lignocaine} > \text{Amethocaine} > \begin{matrix}\text{Diethoxin}\\ \text{Procaine}\end{matrix} > \text{Tutocaine} > \begin{matrix}\{\text{Carbocaine}\\ \{\text{Hexylcaine}\\ \text{Amylocaine}\\ \text{Piperocaine}\end{matrix}\} > \begin{matrix}\text{Butethamine}\\ \text{Amplicaine}\\ \text{Cinchocaine}\end{matrix}$$

As adrenaline enhances nerve block, all the solutions mentioned below are assumed to contain a concentration of 1/300,000 unless stated to the contrary.

Two per cent lignocaine and 1/300 amethocaine were the only solutions which produced an epidural anaesthesia comparable in intensity to intrathecal spinal anaesthesia. The duration of anaesthesia is just under 2 hours for lignocaine from the time of full onset. For intrathecal lignocaine Adams (1956) estimates the duration to be 2 hours, but the author (Stout, 1958) found the average expectation to be about 70 to 80 minutes from studies of recession time in 100 cases of light and heavy lignocaine. The incidence of signs of absorption with 2 per cent lignocaine extradurally confirms the general preference for a 1 to 1½ per cent solution owing to its lower dosage and presumed greater safety. One per cent lignocaine was found to be similar to procaine 2 per cent but inferior to 1/300 amethocaine, the respective durations being approximately 110, 60 and >180 minutes. Whilst this investigation is far from conclusive as regards toxicity, the impression was gained that 1 per cent lignocaine was less dangerous than 2 per cent procaine in respect of hypotension; it gave much better motor block and of the two solutions there is little doubt that lignocaine provides better operating conditions. The favourable points of 1/300 amethocaine have already been referred to, particularly a greater duration and equivalent, if not better relaxation than lignocaine. Its superiority to procaine mentioned by Bromage (1954a) was fully confirmed.

Diethoxin was found to be a powerful drug next to lignocaine and amethocaine in potency but the incidence of toxic stimulation of the central nervous system was high for such a small series of cases (80).

Piperocaine, butethamine, amylocaine and Amplicaine seemed to offer no particular advantages over lignocaine, amethocaine and procaine.

Tutocaine, a drug which is sometimes classed with tropacocaine and amylocaine as a toxic, out-moded spinal anaesthetic, gave a most interesting differential block. Sparing of motor fibres occurred to a degree not exhibited by any other drug, the sympathetic block was good and toxicity of the 1 per cent solution appeared low. In eight cases when the patient was comfortable

the surgeon complained of absent relaxation during surgery.

One per cent Carbocaine was compared with 1 per cent lignocaine and it was weaker, gave less motor paresis, and appeared to last a shorter time. However, the mean dose of Carbocaine was only three-quarters that for lignocaine and, further, there was practically no toxic effect in the twenty test cases. Ekenstam et al. (1956) in a series of 135 caudal blocks with Carbocaine free of adrenaline found a longer duration than for lignocaine, otherwise the two drugs were similar. It may be that adrenaline gives a greater potentiation with lignocaine than Carbocaine.

The finding of Blundell et al. (1955) that hexylcaine is more powerful than lignocaine was substantiated as far as the 1 per cent solutions without adrenaline were concerned, but with adrenaline there was no question of the superiority of lignocaine.

It was confirmed, of course, that cinchocaine is the longest acting of the drugs tested, but its blocking action was much feebler than 2 per cent procaine and only slightly more powerful than 1 per cent procaine, the weakest of all the solutions used. As the cinchocaine used here might have deteriorated with storage in the heat, the 1/600 solution was diluted with distilled water and used as a 1/1500 intrathecal anaesthetic by the method of Howard Jones (the saline was ⅓N instead of ½N). Anaesthesia identical with the well established response to "Light Nupercaine" was obtained. The 1/300 solution, the strongest used, would probably not be regarded as safe by most authorities on local anaesthesia, but even then only 7/20 unsupplemented anaesthesias could be obtained. However, its use in conjunction with light general anaesthesia for prolonged hypalgesia would be unsurpassed.


## SUMMARY

The criterion of toleration of surgery without supplemental anaesthesia was used in an attempt to evaluate relative potency of twelve local anaesthetics in epidural analgesia by samples of twenty cases with each of thirty-five solutions. The two most powerful drugs were lignocaine and amethocaine. Signs of systemic absorption such as coma and generalized analgesia were prominent with lignocaine. 1/300 amethocaine gave a better block than 1 per cent lignocaine. There was a most marked difference in potency between 2 per cent and 1 per cent procaine and hypotension seemed to occur more often with procaine than with other drugs. Two per cent procaine gave a better sensory block than 1 per cent lignocaine, but a very much weaker motor block. Carbocaine appeared slightly weaker than lignocaine but less toxic. Cinchocaine gave a weak block but of longest duration. Tutocaine showed a selective block of sensory fibres. The addition of adrenaline greatly increased the potency of nine of the twelve drugs and reduced the variation in spread of the other three.

There was a tangible increase of intensity of nerve block with age. Illustrating the effect of gravity on lumbosacral penetration, the maintenance of the sitting position for 5 minutes after lumbar injection gave a 60 per cent chance of sacral hypalgesia and a 20 per cent chance of analgesia equivalent to the lower thoracic zone. The use of a viscous solution with preliminary saline plombage gave only slight limitation of spread compared with a nonviscous preparation.



## ACKNOWLEDGMENTS

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## BOOK REVIEWS

*Selected Scientific Papers and Addresses* (1st edition). By Ralph Waters, M.D., Published by Richard Forreger and Forreger Co. Inc.

To read through these papers of Dr. Waters is to live one's own anaesthetic life over again. With the exception of cyclopropane, which Waters introduced into practical anaesthetics himself, there is no matter in these papers which was not a subject of serious consideration among British anaesthetists. The open airway endotracheal anaesthesia, oxygen want, acapnia and other problems connected with $CO_2$, spinal anaesthesia, artificial respiration—these are the subjects with which Waters deals and with which our daily life was intimately concerned.

These various addresses give me the impression that Waters got more co-operation from the surgeons with whom he worked than we did in this country. Once when I took a sphygmomanometer into the gynaecological theatre the gynaecologist asked what it was, and when I told him, he said, "We want none of your —— science here." Though that was a somewhat forcible way of expressing it, characteristic of the man, it did represent the attitude of surgeons to anaesthetists during the period covered by these addresses of Dr. Waters. In my own experience there was one great exception to this sweeping condemnation, Arthur Henry Burgess. He was interested, enthusiastic, patient, and helpful.

Contemporaries of Waters already familiar with the scientific problems with which he deals will find still further interest in the human side. Should nurses give anaesthetics? Should anaesthetists collect their fees or should surgeons do it for them? How to organize an efficient anaesthetic service? These questions have a very familiar sound. Largely due to the establishment of the Association of Anaesthetists they have been satisfactorily answered in this country. The formation of this association was the work of British contemporaries of Waters and the enhanced prestige and professional and economic position of the present-day anaesthetist is largely the result of their labours, an insight into which Waters gives so excellent an account. As this is only a first edition we may expect to have more of them. This is well, because apart from an address on Snow there is no reference to chloroform, even though Waters wrote a book on the subject.

*Fluid Balance Without Tears, or The child's guide to electrolytes* By G. L. Bunton, M.Ch., F.R.C.S. Price 4*s*. 6*d*.

This small pamphlet deals with the body's balance sheet with regard to water and certain salts, mainly sodium and potassium chloride, and the changes that frequently ensue after surgical operations and pathological conditions involving abnormal losses of these essential constituents. Mr. Bunton says in his preface that it is "for the guidance of students and housemen"! It is certain that there will be fewer occasions for tears if these students and housemen follow the advice given than if they fail to do so. It is perhaps too much to expect that there will be no tears. The second part of the title, *The child's guide to electrolytes*, suggests that the dose of precocity normal to each rising generation as it comes along has been somewhat unexpectedly enlarged. It is for these that the text is illustrated, presumably.

*E. Falkner Hill*

*Brit. J. Anaesth.* (1957), **29**, 512

# THE POTENCY OF LIGNOCAINE SPINAL ANAESTHESIA

BY

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LIGNOCAINE has as yet been very little used for spinal anaesthesia, Adams (1956) alone having reported a series of cases in this country. This is probably because at the present time general anaesthesia is the rule and there is little interest in finding a better spinal analgesic.

Having been very impressed by a sample of 5 per cent hyperbaric lignocaine (Xylocaine), it was decided to attempt a controlled comparison with 1:200 cinchocaine (Nupercaine) in order to assess its potency. Preliminary trials with 1 per cent and 2½ per cent solutions had not proved rewarding.

## METHOD

Thirty unselected male cases of bilateral inguinal hernia, aged from 26 to 65 years (mean 50 years), were operated upon under spinal anaesthesia by the same surgeon and anaesthetist in the same theatre, the first side being done under 5 per cent lignocaine (sp. gr. 1018) and the second, a week later, under 1:200 cinchocaine (sp. gr. 1024).

Premedication was with papaveretum 20 mg and hyoscine 0.4 mg given 1½ hours pre-operatively, those over 60 years of age receiving morphia 10 mg and atropine 0.6 mg 1 hour pre-operatively.

The patient was placed in the lateral position on the operating table in the anaesthetic room, lying on the side to be operated upon. The table was tilted so as to give the vertebral column a definite cephalic tilt from the horizontal. Lumbar puncture was performed in the third lumbar space and 1.5 ml of the selected drug was injected slowly through a 22 gauge needle. On completion of the injection the patient was immediately turned on to his back and the thighs flexed on the abdomen for about 1 minute, whilst 15 mg of methylamphetamine were injected both intravenously and intramuscularly. The patient, having been blindfolded was asked to raise his legs, and only if there was no paralysis was a sensory test made before wheeling him into the theatre.

During towelling up his reactions to the insertion of the towel clips just above the operative field were watched. Patients were questioned about sensations during the various procedures such as skin incision, muscular, cord and sac traction, but no sensory levels were tested until the operation had been completed, when the degree of paralysis of the legs was also assessed.

## RESULTS

The height of anaesthesia produced by lignocaine and cinchocaine respectively is shown in the diagram. Each of the 30 cases is represented by a pair of lines, the continuous line representing the action of lignocaine and the interrupted one that of cinchocaine. The upper limit of each line represents the level below which there was anaesthesia to pinprick and the lower limit of each line the level below which the sense of touch was eliminated.

The sense of touch was abolished in 28 out of 30 cases with lignocaine as against only 10 with cinchocaine, whilst with lignocaine it was frequently abolished as high as the operative field.

Lignocaine produced analgesia and the abolition of the sense of touch about two and four segments of the cord respectively higher than did cinchocaine. This was probably due to the diffusion of lignocaine in effective concentration to a higher level than that of cinchocaine.

In those cases in which the sense of touch was abolished in the operative field traction could still be felt, though not localized.

An unpleasant "wooden" numbness was complained of by 5 patients who had lignocaine.

The duration of anaesthesia and paralysis was

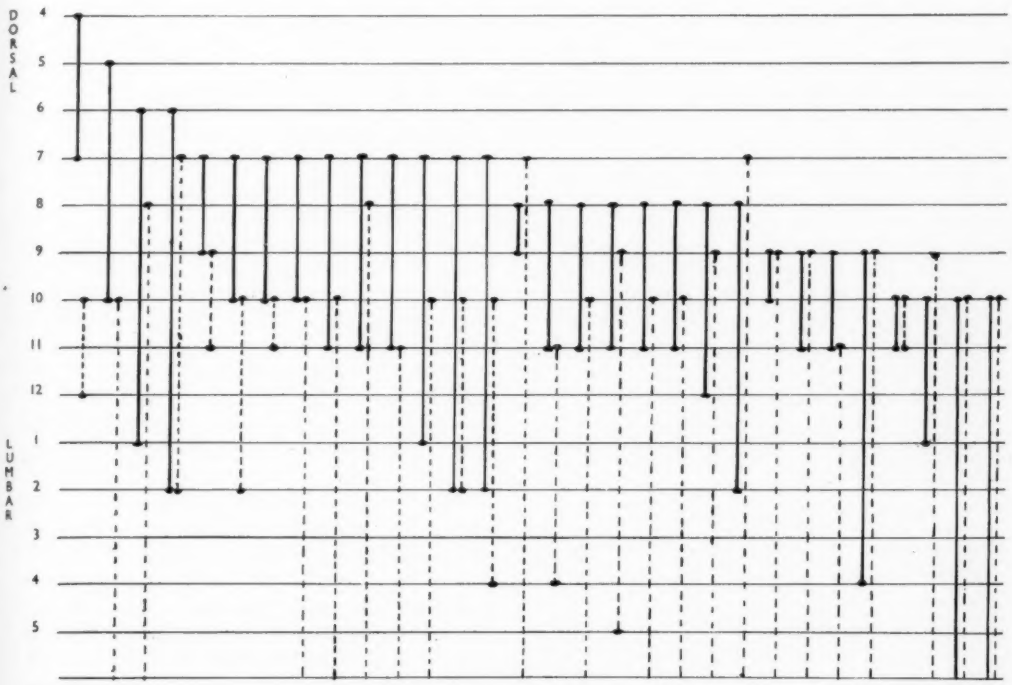


*Diagram illustrating the height of anaesthesia produced by lignocaine and cinchocaine respectively in a series of 30 patients.*

The continuous lines represent lignocaine (Xylocaine) and the paired dotted lines cinchocaine (Nupercaine). The top of each line represents the level below which there was absence of pinprick sensation and the bottom of each line the level below which the sense of touch was abolished.

similar with the two drugs, but the onset of paralysis was much more rapid with lignocaine.

Paralysis of the legs was complete in 29 cases with lignocaine as against only 13 with cinchocaine.

In the series 18 patients preferred lignocaine, 4 cinchocaine and 8 were indifferent.

There was only one case of headache and this occurred with both lignocaine and cinchocaine.

## CONCLUSIONS

From these observations it would seem likely that 5 per cent lignocaine is a more potent spinal anaesthetic than 1:200 cinchocaine and in many cases eliminates the sense of touch. It produces anaesthesia to a higher level by means of diffusion.

The only disadvantage encountered with lignocaine was an unpleasant "wooden" numbness which was complained of by 5 in the series of 30 cases.

## ACKNOWLEDGMENTS

I wish to thank Mr. K. H. Taylor for permission to make these investigations on his patients; Sister Nicholson of Stoke Mandeville Hospital for observing the duration of analgesia and paralysis; and Messrs. Duncan Flockhart and Co. for supplies of Xylocaine.

I am greatly indebted to Dr. A. G. S. Hill for much advice in the preparation of this paper.

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# AN UNUSUAL COMPLICATION OF EPIDURAL ANALGESIA

BY

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EPIDURAL analgesia has been advocated by many authorities, Dawkins (1945), Bromage (1954), Hewer and Lee (1957), as a safe alternative to spinal analgesia, because it is singularly free from complications, the main serious ones being inadvertent subarachnoid injection or toxic reaction to absorption of the drug (Bromage, 1954).

We feel, therefore, that the following case report is of some interest.

## CASE HISTORY

Patient aged 37; pre-operative blood pressure 140/70 mm Hg; Hb 74%; complaint, menorrhagia; premedication, papaveretum 20 mg, atropine 0.65 mg; operation, hysterectomy; anaesthetic, epidural block plus light general anaesthesia.

*Conduct of Anaesthesia.*

With the patient in the left lateral position, the epidural space was sought using Salt's needle and extradural space indicator (Macintosh, 1953) introduced in the L3–L4 interspace. The epidural space was found at the first attempt and a test injection of 5 ml of isotonic 1.25 per cent lignocaine with adrenaline 1 : 120,000 (0.25 ml 1 : 1000 adrenaline in 30 ml lignocaine) made. After 5 minutes no signs of spinal block were present and a further 25 ml lignocaine was slowly injected. The patient was then turned on to her back and 400 mg of thiopentone followed by 75 mg of suxamethonium chloride were injected. Intubation was carried out and a 20° Trendelenburg tilt instituted; 20 mg methylamphetamine hydrochloride were injected intramuscularly.

General anaesthesia was maintained with nitrous oxide and oxygen, spontaneous respiration returning within approximately 5 minutes. After an initial fall to 100 mm Hg systolic the blood pressure returned to 120 mm Hg and was maintained at this throughout the operation, which lasted 45 minutes and was completed uneventfully from both surgical and anaesthetic points of view. The Trendelenburg position was maintained throughout, the patient being supported by shoulder rests.

After operation, the foot of the bed was raised until it was obvious that the blood pressure was remaining stable.

Very shortly after regaining consciousness, the patient complained of severe pain in the abdomen and base of the spine. This was looked upon as normal postoperative pain and was treated with morphine, to which it responded. The next day she still complained of pain in the back which again responded to morphine.

On the second postoperative day, she was seen by one of us (B.W.) and was complaining of severe pain radiating from the base of the spine, right up to the back of the neck, and of pain on moving her head. On examination, she showed increasing tenderness from the site of puncture to the base of the skull along the line of the vertebrae. Her neck muscles were also tender. There seemed no loss of movement or sensation of arms or legs and Kernig's sign was negative. Movement of the head caused pain but there was no true neck rigidity. The pain was unaffected by position, but any movement from one position to another exaggerated the pain. There was no pyrexia at this or any other time and no antibiotics were given.

The pain seemed severe enough at the time to warrant another injection of morphine. On the basis that this might well be after pain due to the suxamethonium (Churchill-Davidson, 1954), 2 tabs. codeine co. four-hourly were prescribed. The pain was still present the next day, although it seemed to be reasonably well controlled by the

codeine tablets. The following (i.e. the fifth) postoperative day, the pain was more severe and the patient also complained of headache, although this was not as severe as the backache. By the next day, she felt much better and was very anxious to be allowed out of bed. With some misgiving, as we still considered that the suxamethonium was implicated, we decided to allow her to sit up in a chair. This resulted in a vasovagal attack and she was promptly returned to bed. The pain waxed and waned through the next few days, and on the eighth postoperative day, when there was still complaint of pain and stiffness, it was decided to see if mephenesin would improve the condition. To this end 1 gram of mephenesin carbamate (Tolseram) t.i.d. was prescribed. The effect of this was negligible and it was stopped after two days. X-ray examination of the entire vertebral column revealed no abnormality.

On the tenth day a neurologist saw the case and reported as follows: hypoalgesia up to the level of T3; slightly increased tendon reflexes (R) leg; slight weakness (R) foot.

In his opinion, this was due to dural irritation caused by the large quantity of fluid injected with subsequent stripping of the dura from the periosteal lining of the vertebral canal. He recommended no active treatment, as he considered the condition would clear up spontaneously.

The complaints of backache gradually diminished and by the twelfth day had disappeared. Mild headaches continued to occur right up to the time of discharge on the seventeenth postoperative day.

## DISCUSSION

A perusal of the literature shows that complications of epidural analgesia are few, and backache and headache are rarely reported. When these complications have been recorded, they were of a very mild nature.

Harger (1941) in his review of epidural analgesia does not mention backache as a complication and found no complications in a personal series of 1,000 cases. Dawkins (1945, 1953) reported no cases of headache or backache in his series. Von Helmig (1954), using epidural injections for the treatment of sciatica, recorded no complications.

Bromage (1952) had two cases of backache out of 355 cases, neither of which lasted longer than 24 hours nor showed any residual effects.

Foldes et al. (1956) found that backache occurred in 3 per cent of their series, only one case being severe enough to require medication, and there were no cases of headache.

Jenicek (1955) gives details of a case which he describes as aseptic meningitis following lumbar epidural block, when, eight hours afterwards, the patient had severe headache, neck stiffness and a temperature of 101°F. Spinal tap gave cloudy c.s.f. with a cell count of 9,600 polymorphonuclear leucocytes, but no organisms were isolated. The patient was put on antibiotics and showed complete recovery in four days with no residual signs. No explanation was offered as to the portal of entry of the chemical irritant into the subarachnoid space.

Lee (1956) has found no similar complications among his series of cases.

In the case reported above, the following aetiological possibilities arise:

(1) *Infection.* This is very unlikely. Many other epidural injections have been made at this hospital using the same technique, the same batch of lignocaine and the same conditions of sterility. Our epidural drum containing the needles, lignocaine and adrenaline is autoclaved, and the contents are not handled except by the anaesthetist who scrubs up and wears sterile gloves and gown. At no time was there pyrexia or true signs of meningismus. For this reason and because of the very painful back, it was felt neither necessary nor desirable to perform a lumbar puncture.

(2) *Spinal puncture.* The possibility of dural puncture at some stage after the injection of the test dose cannot be eliminated, but certainly a large quantity of lignocaine was not injected intrathecally, although a certain amount of c.s.f. leakage might have occurred if the dura had been punctured by the relatively large Salt needle. Accidental thecal puncture with the Salt needle has occurred in three cases without any untoward results. Further, the patient's postoperative symptoms were certainly not typical of a post-spinal condition, backache being the main complaint and posture having no effect.

(3) *Suxamethonium afterpain.* This was our original diagnosis but this would hardly give neurological signs. Further, this would appear to be relatively rare in the nonambulant patient. To our knowledge suxamethonium afterpain of such duration has never been reported—six days being the longest period in the recent series reported by Morris and Dunn (1957).

(4) *Dural and nerve root irritation.* Meningeal irritation due to stripping of the dura was the cause suggested by the neurologist who saw the case, similar effects having been seen by him in patients treated for sciatica by caudal injections of large quantities of local anaesthetic solution. These effects rarely lasted more than one or two days.

(5) *Neurosis.* The patient does not appear to be of neurotic type. On the contrary, she behaved admirably throughout what must have been a very tiresome convalescence from an operation, which, from a gynaecological point of view, was completely uncomplicated and we feel that her complaints must be given complete credence.

## SUMMARY

A case of prolonged backache accompanied by mild neurological signs following epidural anaesthesia is described. Its possible causation is discussed.

In view of the unusual nature of this postoperative course, it has been arranged to see this patient again at a later date, and any further findings of interest will be communicated at that time.

## ACKNOWLEDGMENTS

We wish to record our thanks to Dr. McGillivray, Consultant Gynaecologist at Aberdeen Royal Infirmary, whose patient this was, for permission to publish details of the case; to Dr. T. N. Morgan, Consultant Physician at Royal Infirmary, Aberdeen, for his neurological opinion; and to Dr. H. B. Wilson, Regional Director of Anaesthetics, for his interest in this case and his help in producing this paper.

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*Brit. J. Anaesth.* (1957), **29**, 517

# A VARIABLE PATTERN RESPIRATOR WITH PHASED SAMPLING DEVICE

BY

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*Department of Anaesthetics, University of Bristol*

THIS respirator, used with an anaesthetic apparatus, will produce, within limits, any desired pattern of artificial respiration. A negative phase of any desired proportion can be introduced, or positive atmospheric pressures only may be used.

Provision is made for a small sample of gas for analysis to be extracted regularly at any point in the respiratory cycle.

Artificial respiration is produced by alternate compression and expansion of a concertina bellows of the same type as that used in the Coxeter-Mushin absorber. If no negative phase is desired the bellows can be allowed to fill by the natural collapse of the patient's lungs. The movement of the concertina bellows is produced by an electric motor via a worm reduction gear and a system of levers. The pattern of respiration is produced by altering the speed of the electric motor during the various parts of the respiratory cycle. This control of speed of the electric motor is effected electronically via a simple cam arrangement. Electronic control has the advantage of no heavy moving parts; also the power of the motor is the same at all speeds. The circuit used was described by Sowerby (1949), and is extremely simple (fig. 1). The motor is $\frac{1}{8}$ horse power, d.c., shunt wound, with a maximum speed of approximately 1,400 r.p.m. The supply of electricity to the apparatus must, however, be alternating current. It will be seen that the armature of the motor is in the cathode circuit of a thyratron valve (V1). This is a gas-filled valve capable of carrying a relatively high current, but it will only conduct during the half cycle of alternating current when the voltage on the anode is positive. The valve thus rectifies the current for the armature. The field is supplied continuously with direct current via a rectifier and condenser. During the conducting half cycle the motor will turn. During the other half cycle, when the anode voltage is negative, no current can flow through the valve but the motor will continue to turn under its own momentum. It will thus act as a dynamo and keep the voltage on the cathode almost where it was during the conducting half cycle. If the grid voltage is substantially lower than this, due to the setting of the variable resistors (VR1 or VR2)—the reason for there being two of these will be seen later—the valve will not conduct during the next half cycle, and so the speed of the motor will fall. The speed of the motor will continue to fall until the voltage on the cathode is no longer sufficiently high relative to the grid to prevent the valve conducting during the positive half cycles. When the motor is going sufficiently slowly, depending on the setting of VR1 or VR2, the thyratron will again start to conduct and the motor will again be supplied with power. If a load is placed on the motor when it is going slowly it will slow a little more, the cathode voltage on the valve will fall and the valve will conduct fully, thus maintaining the set speed at any load. With this arrangement there is only the slight disadvantage that the filament of the valve supplied by a separate transformer (not shown in the diagram), must be heated before the anode current is switched on (S1). With the particular valve in use at the moment (a mercury vapour thyratron) preheating is required for eleven minutes. This has not been found to be a nuisance in practice. Other thyratrons with a shorter preheating time are, however, now available.

No attempt has been made to make the apparatus explosion proof as the author believes that the only way to avoid explosions is to avoid explosive agents.

If reference is made to figure 2 the general arrangement will be seen. The electric motor (M) is connected via a pulley and belt to a 25 : 1 worm reduction gear (WG). The maximum final speed of this drive under electronic control is 55 r.p.m., the minimum 5 r.p.m. The output spindle of the worm gear is connected to a system of levers by a crankshaft (CS), and these levers have a movable fulcrum (MF). Thus the stroke volume can be varied within wide limits by turning the screw to which the fulcrum is attached. Also connected to the same output spindle is a series of perspex cams, one of which is shown (C1). These are cut from perspex discs of approximately 5 inches in diameter and $\frac{5}{32}$ inch thick. Bearing on any one of these cams and held to it by a spring, is a light metal arm which is attached to the spindle of a potentiometer (P). In the thyratron circuit (fig. 1) this is the potentiometer VR1. The pattern of respiration depends on the shape of the cam used which can be easily filed or cut. The arm bearing on the cam can be moved along the potentiometer spindle by loosening a thumbscrew, and thus the pattern of respiration can be changed by changing from one cam to another. The overall speed of respiration is controlled without significantly altering the pattern by a second similar potentiometer wired in parallel with the first and hand-operated (VR2, fig. 1).

A positive and negative pressure manometer is placed on top of the concertina bellows to show the pressures in the system. In figure 2 two alternative positions for the fork (F) at the end of the leverage system are shown. The position indicated by the firm line, i.e. between the two collars at the base of the concertina, will cause a negative pressure during expiration. The position indicated by the dotted lines, i.e. under the collars, will merely allow the concertina to fall during respiration and to fill passively. If the vertical spindle up the concertina is unscrewed at its base the whole may be swung outwards and the position of the fork changed.

There is an adjustable expiratory valve on the top of the concertina. The more this valve is screwed down the less will be the negative phase component of respiration and vice versa. The

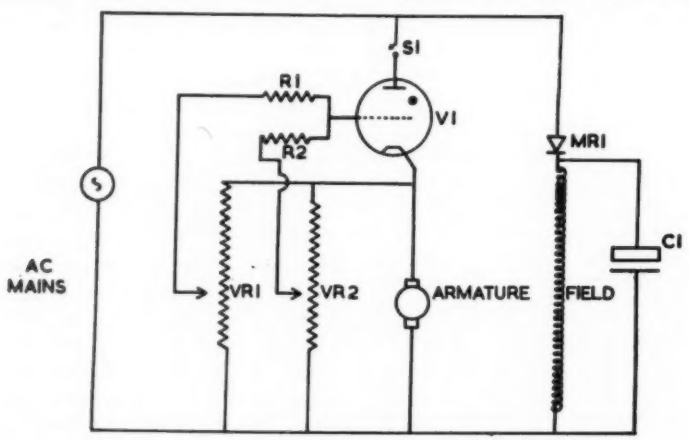


FIG. 1
Circuit of Electronic Control of Respirator.

| | |
|---|---|
| V1. | XG5.500 Mullard. |
| MR1. | Metal rectifier 210 v., 200 m.a. |
| C1. | Electrolytic 16 mfd, 300 v. |
| R1. | 22K $\frac{1}{2}$ watt. |
| R2. | 47K $\frac{1}{2}$ watt. |
| VR1, VR2. | 1 megohm log. |
| S1. | Mains switch. |
| Motor | 210 v. shunt wound d.c., $\frac{1}{4}$ h.p. |

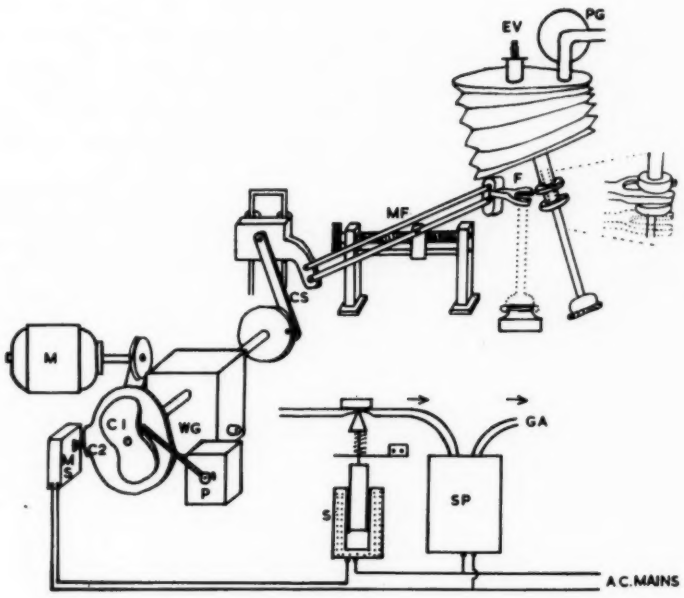


FIG. 2
Diagram of Respirator.

| | |
|---|---|
| C1. | Cam controlling pattern of respiration. |
| C2. | Cam operating sampling device. |
| CS. | Crankshaft. |
| E.V. | Expiratory valve. |
| F. | Fork. |
| G.A. | Gas analyzer. |
| M. | Electric motor. |
| M.F. | Movable fulcrum. |
| MS. | Microswitch. |
| P. | Potentiometer. |
| P.G. | Pressure gauge. |
| S. | Solenoid. A.C. type. |
| S.P. | Gas sampling pump, Mark I, Charles Austen, Ltd., West Malling, Kent. |
| W.G. | Worm Gear, Radicon No. 1, David Brown Gears (London), Ltd. |

Inset shows alternative position of fork. Illustration also shows how spindle supporting concertina bellows may be swung out to change position of fork.

degree of negative phase depends also to a certain extent on the total flow of gases into the machine.

The phased sampling device is actuated by another cam (C2) on the same spindle as C1. This is circular except for a projection $\frac{3}{16}$ inch high, and extending for about 2 inches along the circumference. This projection closes a microswitch (MS). This switches on a small gas sampling pump (SP) and also causes a solenoid (S) to release from a spring clip the rubber tubing coming from the patient. This spring clip prevents respiratory pressures from driving gases into the analyzer at times other than when the pump is working. In normal use the cam (C2) is arranged to close the microswitch at the end of expiration. Gases are taken from the patient's trachea via a Cobb's endotracheal connection, and so a measurement of the patient's alveolar carbon dioxide is obtained. If desired, by rotating the cam (C2) on its axis samples can be taken at any other phase of respiration. Figure 3 shows some patterns of respiration obtainable (with the appropriate cams used). Figure 3A shows a pause at expiration only. Figure 3B shows a pause at both expiration and inspiration, and shows also the effect of the hand speed control. Figure 3C also shows the effect of rotating the cams through 90°. These last two patterns are not recommended, but are included to show what can be done.

No attempt has been made to make the device patient-triggered as it has been used so far only for the completely paralysed patient. However, triggering of the electronic control should present no difficulties if it were desired.

Figure 4 shows the apparatus mounted on a trolley. Underneath is the sampling pump and solenoid and a Brinkman $CO_2$ analyzer. For routine use, however, the British Oxygen Company's colormetric analyzer, using double strength solution (Capel et al., 1956), has been found to be very satisfactory. Over the motor can be seen the case containing the electronic control and the

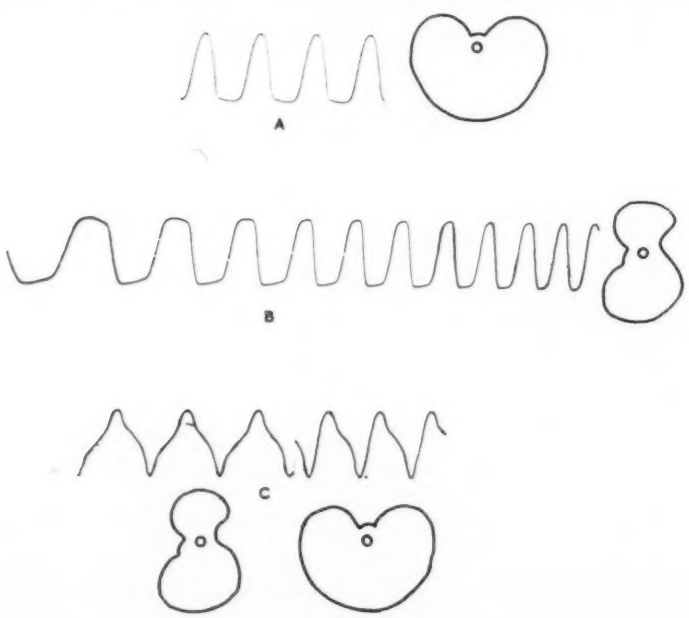


FIG. 3
Patterns of respiration obtainable with outline of cams used to produce them.

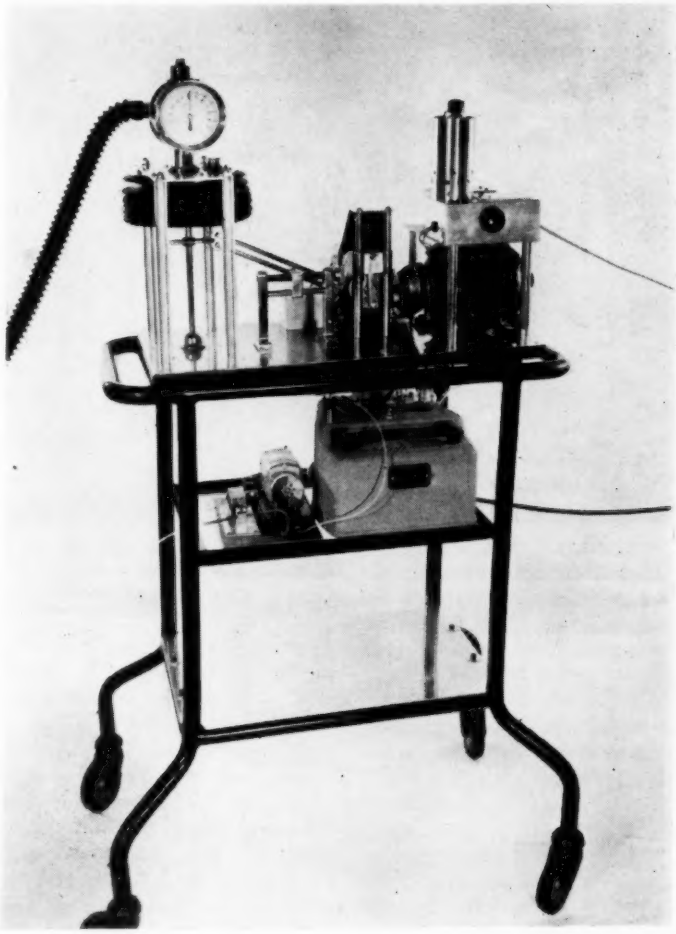

FIG. 4

View of whole apparatus showing on lower shelf sampling device and Brinkman $CO_2$ analyzer.

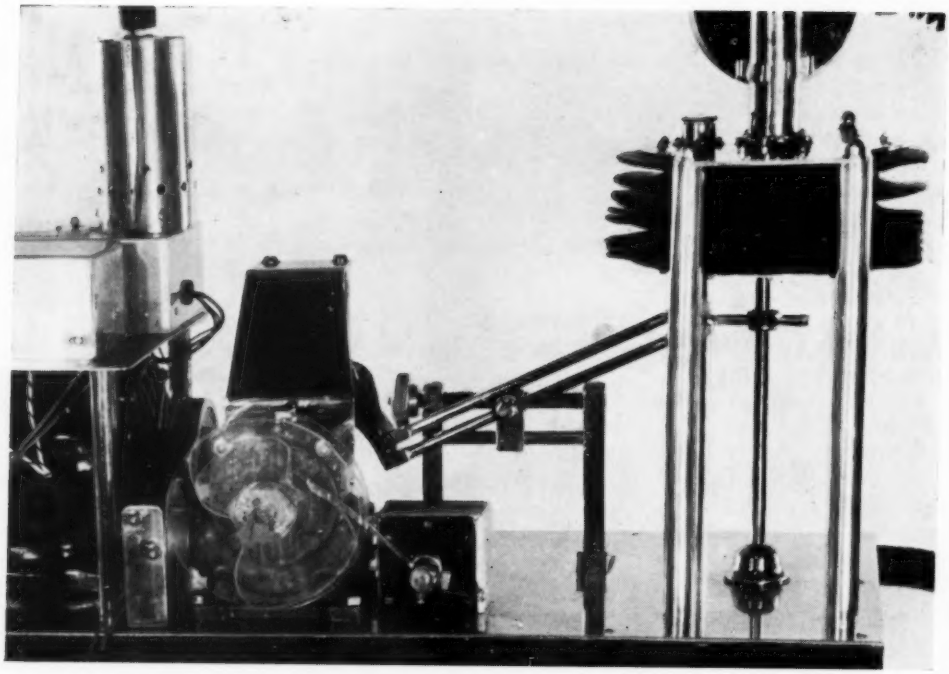

FIG. 5
Back of apparatus showing control cams.

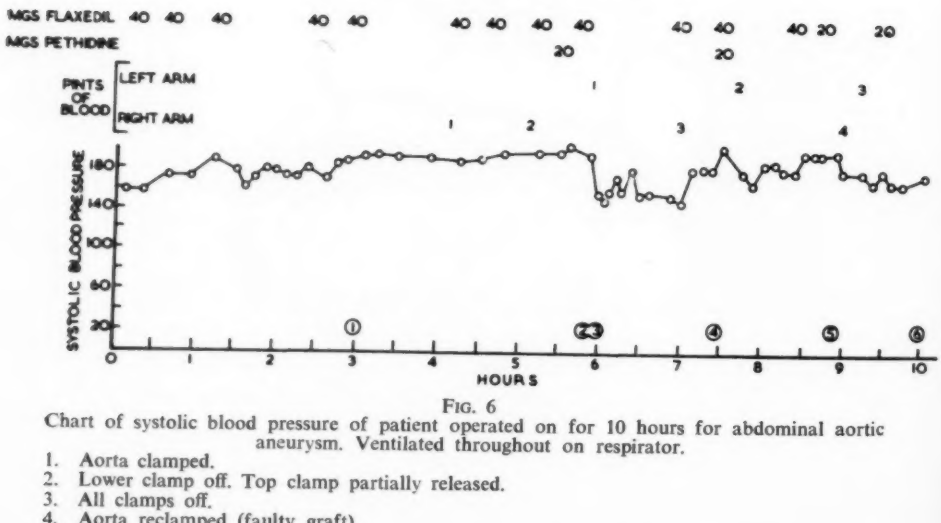


FIG. 6
Chart of systolic blood pressure of patient operated on for 10 hours for abdominal aortic aneurysm. Ventilated throughout on respirator.

1. Aorta clamped.
2. Lower clamp off. Top clamp partially released.
3. All clamps off.
4. Aorta reclamped (faulty graft).
5. Clamps off.
6. Anaesthetic off. Patient conscious. Note very slight fall in blood pressure when aortic clamps were removed.

thyratron valve in a protective cylinder. The black knob is for the overall control of speed of respiration.

This respirator was designed originally for research into the optimum pattern of impressed respiration, and this work is not finished; but the apparatus has been found to be so useful for routine anaesthesia that it was thought that other anaesthetists might be interested to hear of it. Figure 6 shows the chart of the systolic blood pressure of a patient who was ventilated artificially on the machine for 10 hours for a complicated aortic graft. The respiratory pattern shown in figure 3B was used throughout with about equal positive and negative pressures. This patient, a man of 59, appeared to suffer no ill effects from the prolonged period of anaesthesia and relaxation. Blood was replaced as lost. The patient remarked shortly after the operation that he felt far from well; his malaise, however, was shared by the surgical team.

It would seem unlikely that this patient would have been as well as he was after this operation without accurately controlled respiration. The importance of a negative phase in respiration during abdominal surgery where there is likely to be much haemorrhage can hardly be over emphasized (Hubay et al., 1954).

The apparatus was made for me by Mr. G. Meade in the hospital workshop.

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## BOOK REVIEW

*The Principles of Therapeutics*. By Professor J. H. Burn. Published by Blackwells. Price 27*s*. 6*d*.

As the note on the cover states, this book contains the lectures on pharmacology of the basis of therapeutics which are given by Professor Burn to Oxford medical students.

Every anaesthetist who reads this book, and we hope all will, is bound to gain a more intimate knowledge of the working of the human body. The explanation of the part played by acetylcholine, adrenaline and histamine, of which Professor Burn himself has done so much to elucidate, is of course, the work of a master. When we come to the chapter on anaesthetics, however, we begin to wonder with what hospitals, or even if with any, is the professor familiar. Does he really teach his students that "The surgeon makes the injection" (lumbar for spinal anaesthesia) "and having done so then proceeds with the operation"? That must be at least thirty years behind the times. Again he says, "Thirty years ago when the patient was wheeled on a trolley along the hospital corridors and taken fully conscious to the preparation room to be anaesthetized with ether administered on an open mask, this was a frightening experience." It would have been more nearly true fifty years ago; morphia and atropine or atropine alone have been in regular use in some of our hospitals for nearly fifty years, and although this premedication does not render the patient unconscious, its calming effect on mind and body was witnessed to by Ian Mackenzie in these words in 1930. "Preliminary medication marks one of the greatest advances in recent years. Apart from the anaesthetic agent itself there is no single factor in anaesthesia that has afforded the surgical patient more physical and mental benefit and protection than the introduction of the preliminary injection of morphia and atropine or its equivalent." As regards "recent years", he quotes a paper of mine written three years earlier in which reference is made to 20,000 cases in which morphia and atropine or atropine alone were used, so that puts the time back to 1909 when it was first used in Manchester.

*E. Falkner Hill*

# ON CHLOROFORM AND OTHER ANÆSTHETICS

## THEIR ACTION AND ADMINISTRATION

BY

JOHN SNOW, M.D.

*Licentiate of the Royal College of Physicians*

(*Continued from page* 484)

Some attempts have been made to determine, by statistical inquiry, whether the result of operations has been more favourable since the introduction of anæsthesia. These attempts have been confined to the larger amputations, as they are the only operations which sufficiently resemble each other to admit of the application of statistical inquiry; and even in the case of these operations, the inquiries which had been made previously to the introduction of etherization differed widely in their results.

Dr. Simpson took great trouble, in the early part of 1848, to collect the account of the amputations of the thigh, leg, and arm, which had been performed under the influence of ether or chloroform in forty-nine of the hospitals in Great Britain; and for comparison, he collected from thirty British hospitals the results of the same amputations, for the two or three years preceding 1847. He found that the mortality in the period preceding the use of ether was 29 per cent. in these amputations, and the return of patients whose limbs had been amputated under the use of ether and chloroform gave a mortality of 23 per cent.; and he concluded that 6 per cent. of the lives of those who had undergone these amputations had been saved by the use of these agents. In his application for an account of cases, however, Dr. Simpson merely asked for the number of cases, and the number of deaths. Nothing was said as to the time when the amputations had been performed, and there is reason to believe that a number of cases still under treatment may have been included; some of which may have ended fatally after the returns were made. The cutting part of a large amputation is of very short duration. The loss of blood is not great; and patients hardly ever died during the performance of an amputation, but some time afterwards, of diseases which ether and chloroform have not been found capable of preventing. It was therefore not to be expected that these agents should save the lives of so many as 6 per cent. of those who underwent the larger amputations.

In the early part of 1849, I published an account of fifty-five amputations of the thigh, leg, and arm, in which I had administered ether or chloroform, and the mortality was 27 per cent.*

Dr. James Arnott has lately been investigating this subject in a very persevering manner. During the last three or four years, all the cases of amputation performed in the London Hospitals have been reported in the *Medical Times and Gazette*, together with their results; and Dr. James Arnott has stated that the average per centage of deaths, after the three

* Lond. Med. Gaz., vol. xliii, p. 230.

kinds of amputation above mentioned, is 34.4 per cent. There is obviously no means of ascertaining what was the mortality after amputations in the hospitals of London before the use of ether and chloroform, but Dr. James Arnott brings forward four papers, which state the mortality for a short period, in four hospitals, a little time before the introduction of etherization. The number of cases in these papers is 174, and the number of deaths 41. Dr. James Arnott states the per centage of deaths to be 21.9,* although it appears to me that it would be 23.5; and it may be remarked that the four papers from which these numbers are taken might probably not have been published if the numbers they contain had not been favourable. Dr. James Arnott, in a second table, gives the number of amputations and deaths from the several hospitals in London which are named. The cases are said to have occurred "during eighteen months from June 1855 to June 1856, inclusive;" here being obviously a mistake. The number of cases is 204, and the number of deaths 61; which would give a mortality rate of 29.9 per cent.; almost exactly the same as that which Dr. Simpson had obtained from thirty provincial hospitals before the use of ether. The The numbers in Dr. Simpson's table are 618 cases, and 183 deaths, or 29.6 per cent. Dr. James Arnott does not, however, mention the per centage of deaths in his second table, but gives another table, for a different period, in which the names of the hospitals are not given. The numbers he gives in this table are 430 cases and 148 deaths, giving a mortality of 34.4 per cent. Dr. James Arnott therefore concludes that chloroform causes the death of upwards of 12 per cent. of those who inhale it for the purpose of undergoing amputation. It is evident that he can have had no experience of the effects of chloroform, or he would have perceived at once the error of his conclusions, for this agent is given for a much longer time than in amputations, in numerous operations of which the entire mortality is less than 12 per cent. In the removal of tumours of the female breast, for instance, more chloroform is generally administered than in an amputation, and the loss of blood is also much greater, yet the entire mortality after this operation is considerably less than that which Dr. J. Arnott supposes that chloroform adds to the ordinary mortality after amputations.

Dr. J. Arnott stated that the pyæmia, of which many of the patients died after amputation, had probably been rendered more prevalent and fatal by the use of chloroform; but Mr. Thomas Holmes, in one of his able replies to the remarks of Dr. Arnott, points out that pyæmia had been prevalent also in cases of compound fracture, and others in which the patients had no chloroform. He stated that this complaint had been more prevalent in St. George's Hospital during the three years in which Dr. J. Arnott's statistics of amputations had been collected, than in the previous five years in which chloroform had been used.†

A very valuable paper on the result of operations performed in the Newcastle

* Med. Times and Gaz., 1856, vol. ii, p. 413.

† Med. Times and Gaz., 1856, vol. ii, p. 478.

Infirmary, before and since the use of ether and chloroform, was published last year by Dr. Fenwick.* He says, respecting his tables, "in the first series are included the operations registered in the operation books of the Newcastle Infirmary from 1823 to 1843; but, as the record is imperfect, the actual period embraced is seventeen and a half years. In the second, are the operations registered since the first employment of ether; and as the use of anæsthetic agents has been general in all the more important operations since that time these figures may be used to show any disadvantages likely to arise from the employment of chloroform." Dr. Fenwick continues: —"Before the use of chloroform, there were registered 225 amputations of the thigh, leg, and arm, of which 54, or 24 per cent died. Since the use of anaesthetic agents, 149 cases of similar operations have been recorded, of which 36 died, showing also a mortality of 24 per cent.

"Before, however, we can draw any conclusion from such facts, we must carefully exclude all those circumstances which are already known to produce an effect upon the mortality of amputations. It is, for instance, well known that amputations performed on account of accidents are, on the whole, nearly twice as fatal as those required for long standing disease. Now if we divide the foregoing numbers into these two classes, we shall find that before the introduction of chloroform there were 144 pathological amputations, with a mortality of 19 per cent.; while since its employment there have been only 61, of which 13 per cent have died; and while of 81 traumatic amputations which took place in the former period 32 per cent. died, only 31 per cent perished in the latter period. The equal mortality obtained from a general average of all amputations is thus seen to have arisen from the smaller comparative number of operations performed for diseases."

After giving a table which shows the result of each kind of amputation in the two periods, he adds, "It is plain from the above table that since the employment of chloroform there has been a diminution of mortality; thus in amputations of the thigh for disease there has been 5 per cent. less death, while after accidents 17 per cent. have been restored to health, who formerly would have perished. In the pathological amputations of the leg there is a difference of 8 per cent. in favour of chloroform; and while one out of three died after the removal of the forearm for accidents in the former series of cases, no death had occurred out of eight in the in the latter. The only exceptions are to be found in the traumatic amputations of the leg, and in the pathological amputations of the arm. In the former there is an excess of deaths since the introduction of chloroform amounting to 5 per cent. and in the latter, the cases, being only two in number, do not warrant us in drawing any deduction from them."

* Ib., 1857, vol. i, p. 559.

(*To be continued*)

## CORRESPONDENCE

### ANAESTHESIA FOR ADENOTONSILLECTOMY

Sir,—I would like to offer my congratulations to Dr. Andrew Doughty for his excellent paper entitled "Anaesthesia for Adenotonsillectomy" (*Brit. J. Anaesth.* (1957), **29**, 407). As Dr. Doughty so rightly points out, the child should be entitled to receive the maximum benefits from the improvements in anaesthetic techniques which have taken place during recent years.

I cannot, however, agree with Dr. Doughty's outright condemnation of the oral route of premedication, although I decided some time ago that quinalbarbitone was a too long acting drug to use for the purpose. Having made this decision I began to investigate the use of methylpentynol as a premedicant for children undergoing adenotonsillectomy and have recently completed a series of 500 cases. I did not use the papaveretum and scopolamine combination as advocated by Dr. Doughty because, although I agree that children tolerate this combination well in the correct dosage, some depression of the respiratory centres and cough reflexes must result from the use of these drugs, and such depression I consider undesirable in children undergoing operations upon the nose and throat.

The method of premedication I used in my series was as follows: The child was given methylpentynol (Oblivon Elixir) 1½ hours before operation and atropine sulphate subcutaneously ½ hour before operation. It is important that an oral premedicant is given well before the dose of atropine or scopolamine being used, otherwise absorption from the stomach will be delayed.

Children weighing from 21 to 35 lb received two teaspoonfuls of the elixir (500 mg of methylpentynol). Children weighing from 36 to 55 lb received three teaspoonfuls of the elixir (750 mg methylpentynol), and children weighing over 55 lb received four teaspoonfuls of the elixir (1000 mg methylpentynol). This corresponds very roughly to a dose of 18 mg per pound body weight.

At the beginning of the series all children were anaesthetized by the "standard method"; i.e. ethyl chloride and open ether or nitrous oxide induction followed by nitrous oxide, oxygen and ether insufflated down the side tube of a Boyle-Davis gag. It was then decided to investigate the intravenous technique, using thiopentone for induction followed by suxamethonium chloride, intubation, and maintenance with nitrous oxide, oxygen and minimal ether. At first this technique was used only for the older children and then, as the results proved very satisfactory, it was gradually adopted for the younger children as well.

The series of 500 cases premedicated with methylpentynol and atropine is therefore composed of 325 patients anaesthetized by the ether insufflation technique and 175 patients anaesthetized by the intravenous technique.

Twelve patients of the series arrived in the anaesthetic room asleep and anaesthesia was induced smoothly without the patient awakening. Four hundred and thirty-four patients arrived awake and in an apparently peaceful frame of mind and were quiet and co-operative during the induction, whichever method was used. The remaining 54 patients arrived in an apprehensive state of mind but 39 of these could be reassured; this was a notable feature following methylpentynol, and induction was carried out smoothly. Fifteen patients out of the 500 remained nervous and unco-operative and the effects of the methylpentynol were not considered satisfactory; only 2 of these, however, were being induced by the intravenous route.

Only 2 patients in the series vomited during induction and both these were thought to have done some illicit drinking pre-operatively. Five others made attempts to vomit but this was considered to be due to ether rather than methylpentynol. No patient who was induced with thiopentone vomited or regurgitated stomach contents during induction.

None of the patients gave cause for anxiety during operation, but 5 patients showed some

degree of laryngeal spasm immediately post-operatively 3 of these being induced by inhalation and 2 intravenously and intubated. All responded to pharyngeal toilet and posture.

Thirty-five patients were unduly restless post-operatively and it is interesting to note than 28 of these were from the 175 intravenous cases and only 7 from the 325 inhalation cases. The longest case of restlessness lasted 30 minutes.

All cases were examined personally post-operatively and apart from the 5 who developed some laryngeal spasm, mentioned above, all were considered to be very satisfactory. The nursing staff were well pleased with the pre- and post-operative behaviour of these children.

In conclusion, I would wish to agree heartily with Dr. Doughty that the intravenous and intubation technique provides very satisfactory anaesthesia for adenotonsillectomy in children, but would submit that oral premedication with methylpentynol followed by subcutaneous atropine sulphate offers a satisfactory alternative to papaveretum and scopolamine, and does not possess the possible depressant effects of the latter drugs upon the vital centres and reflexes.

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## CONTROLLED RESPIRATION IN NEUROSURGICAL ANAESTHESIA

Sir,—May I express my substantial agreement with the experiences of Dr. Diana Furness using controlled respiration for neurosurgical operations (*Brit. J. Anaesth.*, 1957, **29**, 415)?

I have used continuous controlled respiration by mechanical respirator for all the neurosurgery at Frenchay Hospital for over three years, and have records of about 500 cases.

The technique throughout has been; premedication with atropine; induction with thiopentone; curarization with tubocurarine; intubation with a cuffed tube, and nitrous oxide and oxygen with a circle absorber, but with excess gases and semiclosed circuit. In about one-fifth of the cases, morphine 10–15 mg has been given intravenously.

In view of the duration of operation, it is usually preferable to extra doses of thiopentone. In all cases with papilloedema and/or indications of raised intracranial tension, intravenous sucrose, 50 to 100 ml of 50 per cent solution, has been given immediately after induction and intubation.

The advantages appear to be:

(*a*) Low intracranial tension, with normal arteriolar vascularity and blood pressure. This seems to me important, as the cause of tissue death in cases of raised intracranial tension is vascular ischaemia, as it is in the case of strangulation of a hernia, secondary to venous engorgement. It does not seem sound practice to lower the blood pressure in such cases, often with a pressure already raised in an attempt to compensate for defective cerebral circulation, unless access can be obtained by no other means.

The bloodlessness, and lack of cerebral congestion and swelling, obtained by the technique outlined are as good as that obtained by me with safe degrees of controlled arterial hypotension, and are without the hazards of reactionary oedema, secondary to unmeasurable inadequacy of the cerebral circulation.

Though it has been apparently confirmed that the total circulation through the head is not affected by the use of controlled hypotension, personal observations have convinced me that the cerebral cortex can be made relatively ischaemic for long periods by permitting a low blood pressure, and it is possible that a shunt is responsible for the apparent anomaly.

Bleeding, after all, is in the main from the veins and capillaries, and cerebral decongestion (which is the primary object of craniotomy) is a more logical approach to its control than arterial hypotension.

(*b*) Recovery at the end of surgery is immediate. Neostigmine and atropine are usually given, and the patient is awake, with normal pharyngeal reflexes and tongue and jaw tone, as the tracheal and pharyngeal toilet is performed and the tube withdrawn.

This means that any neurological abnormality then or subsequently can be observed, and one does not have to try to guess how much of a given picture is due to narcotic depression, and how much to pathology or the effects of surgery.

(*c*) Postoperative course. The incidence of postoperative brain swelling is now almost nil. Prior to the use of this anaesthetic technique,

but using all the accepted methods of avoiding cerebral congestion, this serious complication was not uncommon as a sequel to the removal of a large tumour, or even resulted from overlong retraction of a frontal pole. Immunity appears to be due partly to the direct effect of avoiding hypotensive ischaemia in the compressed brain, but principally perhaps to the far better surgery which is possible, when the brain is no longer turgid, and tumour vessels do not pour with blood at a touch.

(*d*) On general principles, the avoidance of having to give volatile anaesthetic agents is an advantage. They all predispose to vomiting to some extent, and those which are not explosive are toxic, even if only to a minor extent. We are often dealing with patients in very poor physical condition. Nitrous oxide, given with adequate oxygen, over the periods of time required, has not been shown to cause recognizable tissue damage. Ether, chloroform, trichloroethylene, cyclopropane, and halothane, all cause, or can cause, cloudy swelling of the liver, and it might be guessed that the central nervous system is not immune.

(*e*) Blood. Blood losses have been reduced, and it is now common practice to remove a large vascular tumour without requiring any blood transfusion, though blood is not withheld where more than approximately 180 ml are lost. With arterial hypotension, induced by arteriotomy or ganglion blocking drugs, we could obtain the same type of operating field, but on restoring the blood pressure there was almost invariably a period of increased capillary and arteriolar oozing, which it would seem safe to conclude is a mild reaction to ischaemia. Blood transfusion, even now, is not wholly without risk, and the avoidance of large transfusions is therefore advantageous.

(*f*) The following are quoted as specific examples of the improvement. Access to the chiasma and pituitary region is always difficult. In order to improve access, we used to tap the ventricles, or to insert lumbar drainage catheters. As a general practice, we no longer do this, and access is very good, without any necessity for heavy retraction of frontal lobes.

If a ventricular needle is inserted, the tension is low, and it may be pointed out that the c.s.f. tension can be raised or lowered by suitable adjustments of the respiratory apparatus.

Secondly, in trigeminal section operations performed in the sitting position, we used to do lumbar punctures, to diminish retractor pressure on the temporal pole in the middle fossa. With controlled respiration, the brain is lax, as though such a lumbar drainage had been carried out, and retraction is easy.

(*g*) Operating time. Our average operating time per case has dropped by at least 30 per cent, simply because long periods do not have to be spent on trying to control oozing, and a tumour can be removed without recourse to the primitive method of "gutting", and removal of the "capsule" afterwards.

Possible hazards:

(*a*) Controlled respiration removes an important indication of the patient's state. In practice this is of no importance whatever, except in operations in the neighbourhood of the medulla. The use of a cardioscope, and adequate attention to the circulatory state is sufficient to ensure that controlled respiration is being correctly applied. Excessive ventilation leads to peripheral pallor, some bradycardia and mild hypotension; in children and infants, overventilation is easier, and it is possible to produce dangerous degrees of circulatory depression. On the other hand, inadequate respiration produces the signs of catabolite retention and, of course, congestion. In the absence of full curarization it also leads to minimal respiratory movements. In practice, full curarization is not carried out, and this appearance of small diaphragmatic movements can then be used to control the respiratory minute volume.

(*b*) Persistent curarization. A good deal has been written about this. I have been very fortunate, in that I have not so far encountered a case. I do not use suxamethonium for intubation if tubocurarine is to be used. Should the situation occur in a neurosurgical case controlled respiration until recovery occurs is preferable to risking periods of mild asphyxia while attempting to restore respiration; carbon dioxide should never be used.

(*c*) Neostigmine. Occasionally bradycardia and circulatory depression can occur after giving the usual dose. If these patients are not treated,

they may proceed into genuine circulatory collapse. A ward check about 20 minutes after they have left the theatre will enable oxygen and further atropine to be given if required. In our early cases, we had one or two instances of patients requiring urgent blood transfusion for circulatory collapse. Since this possibility has been noted, we have had no further trouble.

(*d*) Subdural haematoma. The low intracranial tension obtainable in sitting patients by this technique may be considerable, and the risk of tearing anastomotic veins must be borne in mind. The principle should be to avoid excessively low tensions by suitable respiratory adjustment. We have not had a case of this complication, but the possibility exists, just as it does when ventriculography or encephalography with air is carried out in the elderly, and the tension is allowed to fall.

(*e*) Air embolism. We have not experienced this. It has been much feared by our surgeons, though with little reason. Two examples should be mentioned. In one, a mastoid emissary vein was cut while a posterior fossa was being approached in the sitting position. The vein did not bleed, but gaped empty. The respirator was stopped immediately in compression, and the vein clipped. No air appeared to have been aspirated, and no ill result followed. In the other, a trigeminal section, the cavernous sinus was inadvertently opened. Inside, the blood was seen to occupy a level above which was air. As each respiration occurred, the level rose and fell slightly.

The respirator was adjusted to a slightly more positive mean tension, and the sinus began to bleed. It was then closed by sutures. No ill result followed. It is very unlikely, of course, that a little air rising into the sagittal sinus will impede blood flow in any way, and it will be absorbed.

If the entry of air is suspected, the patient's head should remain elevated for some hours postoperatively. As this is our usual practice anyway, special treatment does not arise.

Aspiration of air into the great thoracic veins and heart is the danger, and it is fair to say that this is less likely to occur under properly controlled respiration than with spontaneous respiration, because the sudden inspiratory gasp of large volume is impossible.

I was glad to observe from Dr. Furness's letter that she too had some difficulty in convincing her surgical colleagues that the use of a respirator was not just a method of diminishing the anaesthetist's work. However, our own neurosurgeons are now convinced that the technique represents a genuine advance.

Indeed a paper was read by myself on the subject of controlled respiration in neurosurgery before the Society of British Neurological Surgeons at their November meeting last year, in which I attempted to give a more detailed account than is possible here. There is no question in my own mind that we are doing better neurosurgery now than we were prior to 1953, and that this is due to a great extent to the method of anaesthesia which has been practised since that time. I abandoned using arterial hypotension, for any purpose whatever, three years ago.

We are fortunate in possessing respirators for infants and young children, and our collection of cases includes a large number of infants and children, all of whom have been anaesthetized using controlled respiration throughout.

I do not agree with Dr. Furness's conclusion that it is possible to produce equally good operating conditions with " perfect " open circuit anaesthesia. I spent about four years trying, and during that time we used hypotensive anaesthesia on selected cases only. In noncompressed cases, she is undoubtedly correct, but there seems no doubt that controlled respiration can reduce cranial compression in already compressed cases, to some extent, and the best possible classical anaesthesia cannot do this.

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## EFFICIENCY OF THE CUFF ON ENDOTRACHEAL TUBES

Sir,—A patient deeply unconscious after ingested poison had a cuffed endotracheal tube in situ for some 24 hours before death. At postmortem a considerable quantity of bile-stained fluid was found in the air passages; and it was

thought desirable to test the suggestion that, because of its low surface tension, bile can seep past an inflated cuff.

The trachea was removed from a fresh female cadaver and a No. 8 cuffed Magill tube inserted. The cuff was inflated with air as in clinical practice. The preparation was suspended vertically and a few ml of bile from a fresh cadaver deposited above the cuff. After 24 hours the preparation was inspected, when it was seen that the bile above the cuff had not evaporated. The preparation was then inverted and after deflating the cuff the endotracheal tube was removed. The trachea was now opened longitudinally with scissors, when a clear line of demarcation showed that in this experiment the inflated cuff had afforded adequate protection. A control experiment, using saline coloured with methylene blue, gave a similar result.

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## BOOK REVIEW

*Anatomies of Pain.* By K. D. Keele, M.D., F.R.C.P. Published by Blackwell, Oxford. Price 27*s.* 6*d.*

Pain has become very popular of late, only let me hasten to add as a subject to investigate and write about. Earlier this year Masson et Cie published *La Douleur et les Douleurs,* a volume of some 340 pages consisting of essays by different authors on the various aspects of pain. A review of this collection appeared in the May number. Dr. Keele traces the evolution of the ideas that early man held of the nature of pain, up to those of the present day. To primitive man pain was due to the entrance of some object or spirit from the outside world, a demon, the spirit of a dead man seeking a new body, an ill disposed deity, etc. Treatment was directed to placation, exorcism, in other words psychological treatment. Today the psychiatrist has taken the place of the magician. Are we to consider this as a throw-back or progress or as you were? To the Chinese and the Hindu who had become aware of the heart and vessels pain was due to too much heat and was cured by acupuncture. The Greeks were divided. Plato thought that sensation was due to movement of atoms along veins, to the tripartite soul situated in the liver the heart and the brain. Aristotle thinks: "The motions of pleasure and pain, and generally all sensation plainly have their source in the heart and find in it their ultimate termination." The brain he regards as a kind of cooling apparatus or radiator to dissipate the heat arising from the heart. Absurd as we now think the physiology of Aristotle, there are those today who appear to agree with him. The Indian Chief discussing psychology with Dr. Jung said: "I know you white men think with the brain. That accounts for your shortcomings; we red men think with the heart." And Leriche, not satisfied that our carefully mapped out pain paths are entirely adequate to meet the demands of clinical practice, says: "Why should we not believe those who tell us 'My heart pains me'?" So Leriche believes that renal pain is felt in the kidney and cardiac pain in the heart. So we are back to Aristotle. The next step in the history of the development of the ideas about pain is so important, is such a stride, that it can only be likened to coming out of darkness into light. Herophilus described the cerebral hemispheres, the venous sinuses, the cranial and spinal nerves, and showed that the section of one led to paralysis and of another to loss of sensation. Erasistratus seems to have been associated with Herophilus in these discoveries and suggested that man's intelligence was due to the richness of his cerebral cortical convolutions; an opinion scorned by Galen "since donkeys also have very complex brains". The views of Herophilus and Erasistratus suffered eclipse for some 400 years as the Romans would have none of them. Galen brought them into prominence again and by his experiments on the spinal cord did much to confirm and extend them.

He made no attempt to locate the soul, but linked it with such bodily states as intoxication and alcoholism and so refused to recognize its immortality. Dr. Keele adds that this "may in part account for the submersion of Galen's work as a whole for over 1,000 years", owing to its unpopularity with the fathers of the Christian Church! If unhappily we are in for another "dark age", it will be due to the organization of mass suicide upon which we seem now to be engaged rather than opposition between Church and science, for our scientists have become affected by religion and our clergy by science.

Since Herophilus discovered the difference between motor and sensory nerves in 300 B.C., though advances had been made in some departments of physiology, notably the circulation of the blood by Harvey, little progress had been made in the evolution of the ideas on sensation or the paths by which, or the destination to which, sensory impressions made their way, till Charles Bell wrote his epoch-making pamphlet in 1811, *Idea of a new Anatomy of the Brain.* Bell distinguished the sensory root from the motor and traced the sensory tract to the cerebrum. He made the following observation which contradicted and corrected ideas that had been current for hundreds of years. "The sensibilities of the living frame are appropriate endowments, not qualities necessarily arising with life, still less the consequences of the delicacy of texture. Nor can we doubt longer that they are suited to the condition, and especially to the degree of exposure of each part, and destined for the protection of the different organs. We perceive no instance of pain being bestowed as a source of suffering or punishment purely."

Following Bell during the nineteenth century great progress was made in the elucidation of the pain paths. It is crowded with names that every student of physiology is familiar with, even if he is not aware of the precise part played by each. On the clinical side there is Gowers; experimenters are too numerous to mention, but Edinger, Waller and Sherrington come to mind, and Weigert Marchi, Golgi and Ramon y Cajal with their staining methods did much to add to the knowledge and beauty of the c.n.s.

In the chapter on twentieth-century pain mechanisms Dr. Keele gives considerable prominence to Mackenzie's views. He points out that "Mackenzie did not deny that stimuli from viscera produce pain, as is often said, but that the location of such pain is in the viscus." It is good to have attention called to this error for Mackenzie's ideas, though they do not explain many of the pains the clinician is called upon to treat, are fundamentally sound; the replacement of his "irritable focus" by the mechanisms of "facilitation and convergence" being only another method of saying much the same thing. Head's distinction between protopathic and epicritic pain is referred to at some length as is also Trotter's idea on the insulation of the nervous system. The work of Gaskell and Langley brings the autonomic system to the front, and it is at once seized upon as the pathway of elusive and irregular pains who refuse the normal highways of the cerebrospinal tracts. It is in this chapter also that we are introduced to another nervous system—the "reticular," defined as "multisynaptic neuronal paths" as yet somewhat dimly defined, but opened up to investigation by the power of valve amplification and the cathode ray. The centrencephalic system of Penfield and the "visceral brain" of Maclean and Fulton show the growing tendency to centralize the sensorium within the brain, and these with the increasing appreciation of the biochemical aspects of sensation, together with the discovery of the reticular path, constitute the trends of twentieth-century research. *Anatomies of Pain* is the most fascinating book, no review of which, I feel, can do it justice.

*E. Falkner Hill*

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